Journal
OF THE AMERICAN VETERINARY MEDICAL ASSOCIATION

AVMA Convention—Cleveland, August 19-22, 1957

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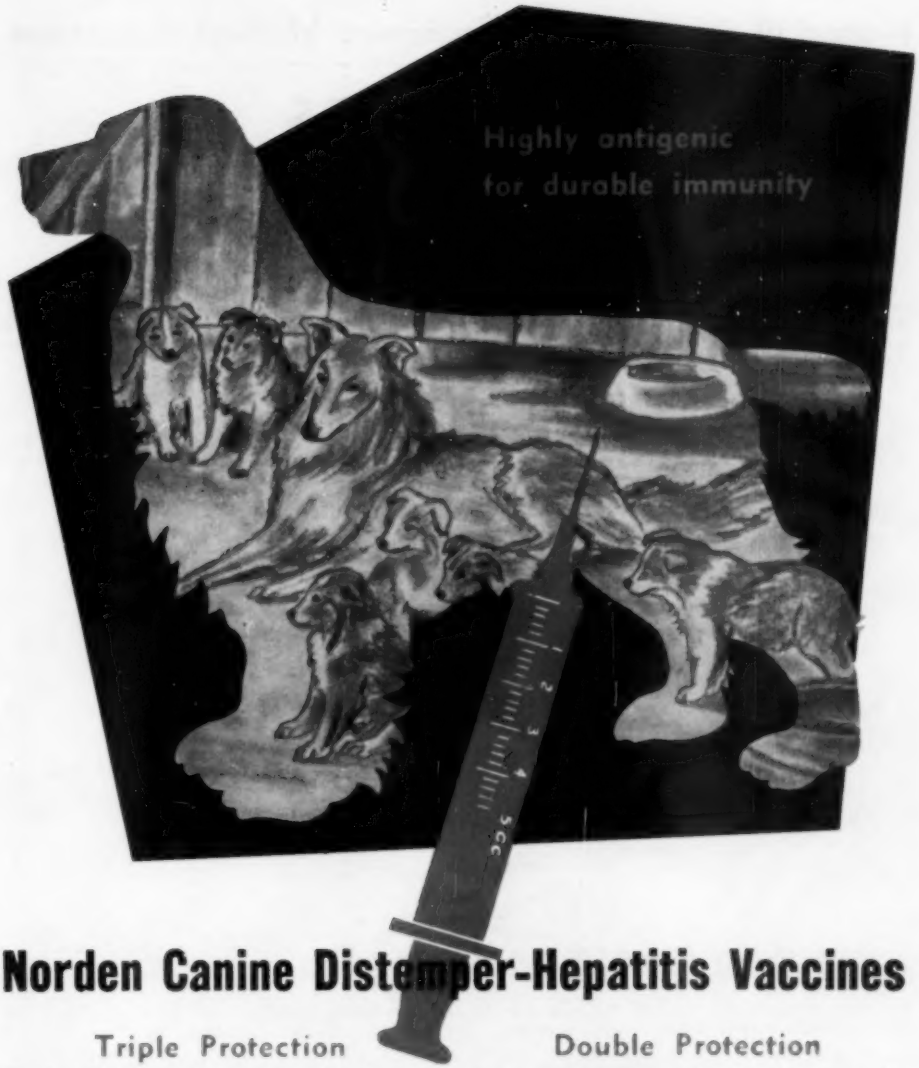

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Correspondence

Dear Dr. Aitken: March 19, 1957

I want to thank you for your letter of March 8 about the abstract (*J.A.V.M.A.*, Dec. 1, 1956: 526) on equine infectious anemia.

It seems that the author of the *German Science Bulletin* misunderstood the statements in the report of Muller and Schützler in *Archiv fur Experimentelle Veterinarmedizin* (vol. IX), which seems to be based on a careful study carried over two and one-half years.

The "anemia-free animals" mentioned in the abstract were a mixed population of horses, some of which might be infected. On page 352 of the original article the authors state: "It is noteworthy that the frequency of 5% (horses with more than 49 typical sublingual hemorrhages among a number of horses without evident clinical symptoms of infectious anemia) corresponds about with the frequency of virus carriers in middle Germany . . . ."

I agree with this and have long realized that it is necessary to ascertain very carefully that a horse population assumed to be virus-free has had practically no chance of contact with infected horses.

If you do that, you will find about 0.3% of anemia-"free" horses with more than 49 typical subclinical hemorrhages, not 5% as is stated in the abstract.

I thought it my duty to help clear up the mystery. Sincerely yours,

s/W. Steck, Bern, Switzerland.

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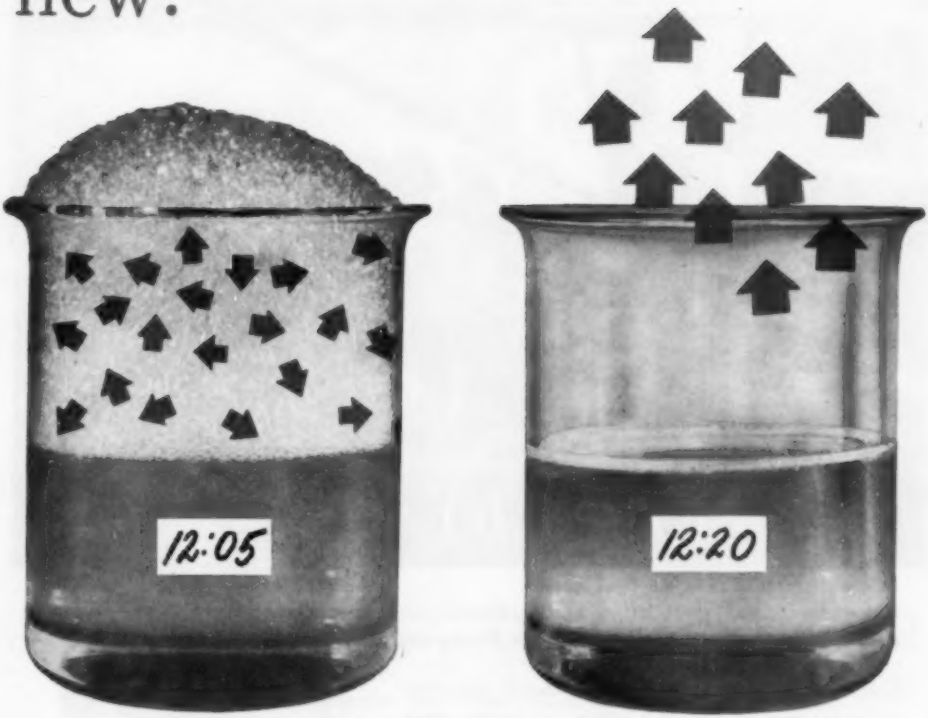

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*Belloff, G. B.: Calif. Vet. 9:16 (Nov.-Dec.) 1956.

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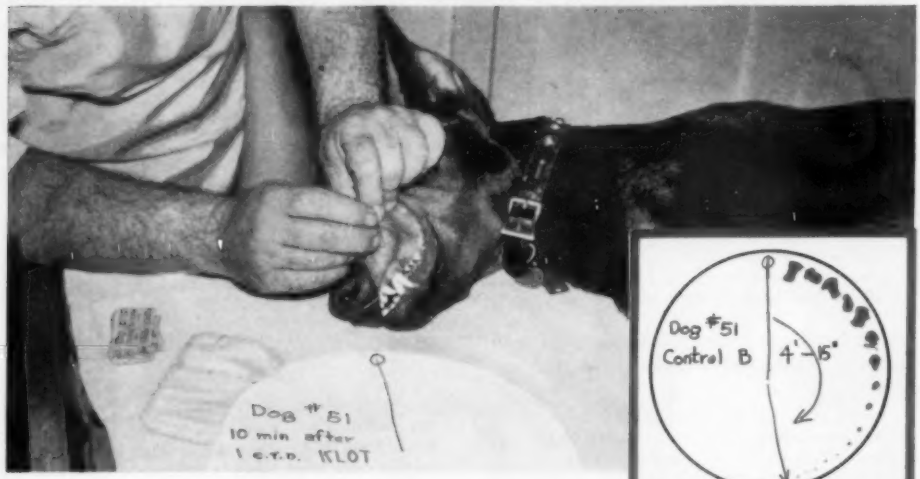

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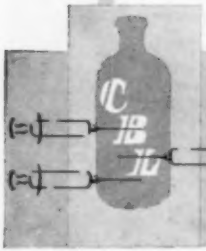

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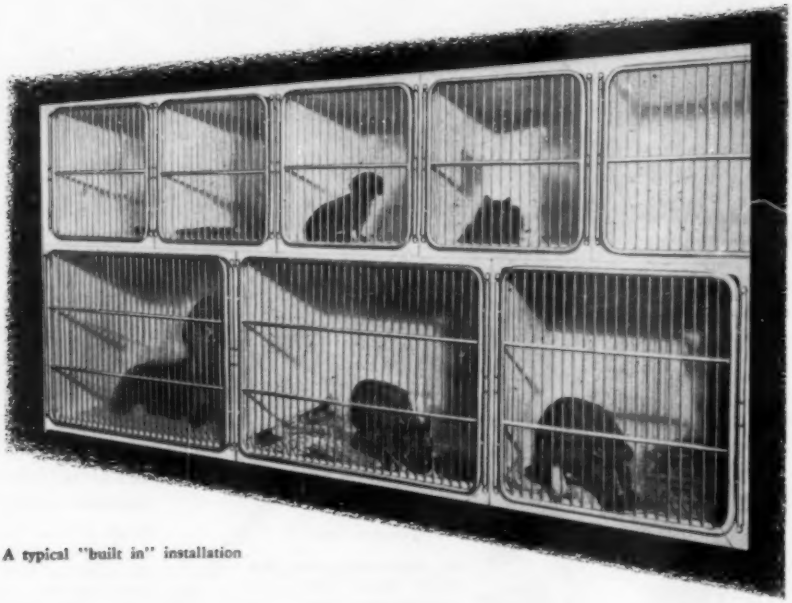

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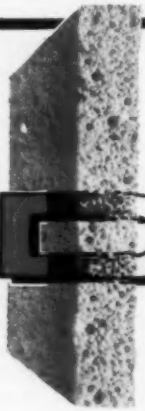

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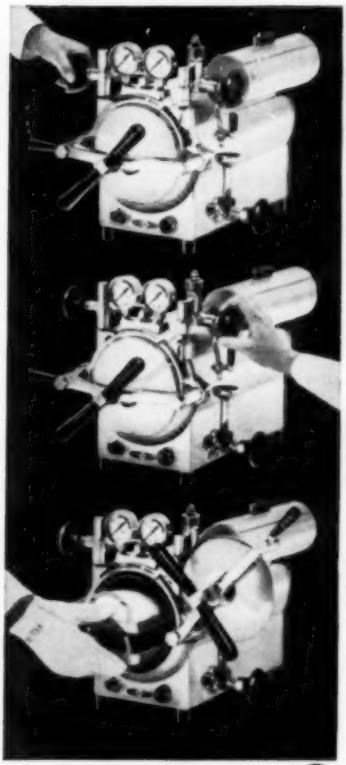

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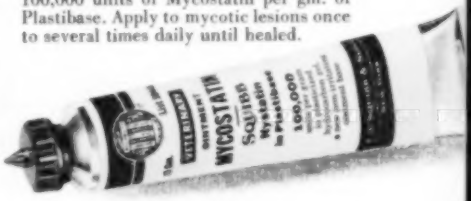

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Packaging: Bottles of 100 and 500.

Anabolin

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DIVISION OF VETERINARY MEDICINE

WARNER-CHILCOTT

Laboratories MORRIS PLAINS, N. J.

News From Washington

Cattle fever ticks were found in Florida on April 23, 1957, for the first time since 1950, when state was declared free of the pests. More than 100 ranches in ten counties were quarantined as of May 16, 1957. A federal-state eradication program is in progress, including inspection and dipping, also tracing movements of cattle and horses shipped out of the area prior to discovery of ticks.

★ ★ ★ ★

Outstanding Veterinary Scientist to Turkey.—Dr. B. T. Simms, ARS, retires from the U.S.D.A., June 30, 1957. He will accept a position with the University of Nebraska for assignment to the University of Ankara (the University of Nebraska has a contract with the latter). Dr. Simms will teach and engage in research at the Veterinary College, University of Ankara, and will leave Washington shortly after June 30.

★ ★ ★ ★

Military.—The Third Army Headquarters, Fort McPherson, Ga., has announced the appointment of John E. Fountain (API, 1956) as first lieutenant, Veterinary Corps, and his order to active duty June 14, 1957, with station at Fort Meade, Md. The Air Force expects to order a limited number of veterinary officers to active duty in July. It is understood that those receiving orders will be from the 1957 Veterinary R.O.T.C. graduates.

★ ★ ★ ★

Legislative Action.—The House passed a bill (H. R. 6548) amending the regular draft act to provide for the procurement of physicians, dentists, and allied specialists. It would replace the so-called doctor draft act, which expires July 1, 1957. This is a committee bill on which hearings were held May 7-8 by the House Armed Services Committee. The bill authorizes special call-up and induction of nonveteran physicians, dentists, and allied specialists to age 35; it continues the national, state, and local advisory committee to the Selective Service System and, among other provisions, continues the annual deferment of . . . preveterinary . . . students at colleges and universities at present levels. Early action in the Senate is expected. In effect, H.R.6548 assures the Armed Forces a supply of physicians and dentists until 1959, through operation of the regular draft, should there be insufficient volunteers for military service.

★ ★ ★ ★

House Committee on Agriculture reported favorably a clean bill (H.R. 6814) to provide for compulsory inspection by U.S.D.A. of poultry and poultry products (see JOURNAL, May 15, 1957, adv. p. 16).

★ ★ ★ ★

Budget requests in U.S.D.A. appropriations, fiscal 1958, for ARS, as passed by the House, were reduced over $15 million, funds for meat inspection being cut $2,132,000. The bill (H.R. 7441) was referred to the Senate Committee on Appropriations on May 16. There is a possibility that the committee might restore all or a part of the cut in meat inspection funds.

★ ★ ★ ★

New Bills Introduced.—Amend Public Health Service Act (H.R. 6874, Rep. Harris, D., Ark.; S. 1917, Senators Smith, R., N.J., and Purtell, R., Conn.; S. 1922, Senators Hill, D., Ala., Kennedy, D., Mass., Neely, D., Wis., Humphrey, D., Minn., and Smathers, D., Fla.) relating to grants for construction of research facilities and authorize grants for medical and dental teaching facilities.

Revise, codify, and enact into law title 21, U.S. Code "Foods, Drugs, and Animals" (H.R. 7100, Rep. Willis, D., La.).

★ ★ ★ ★

Protection against diseases of livestock and poultry (H.R. 6792, Rep. Cooley, D., Ga. (see JOURNAL, April 15, 1957, adv. P. 12, re: companion bill S. 1682).

Social Security amendment (H. R. 7669, Rep. Dingell, D., Mich.) would increase from $4,200 to $6,200 annual earnings in compulsory benefits under Title II of Act and which are subject to social security taxes.

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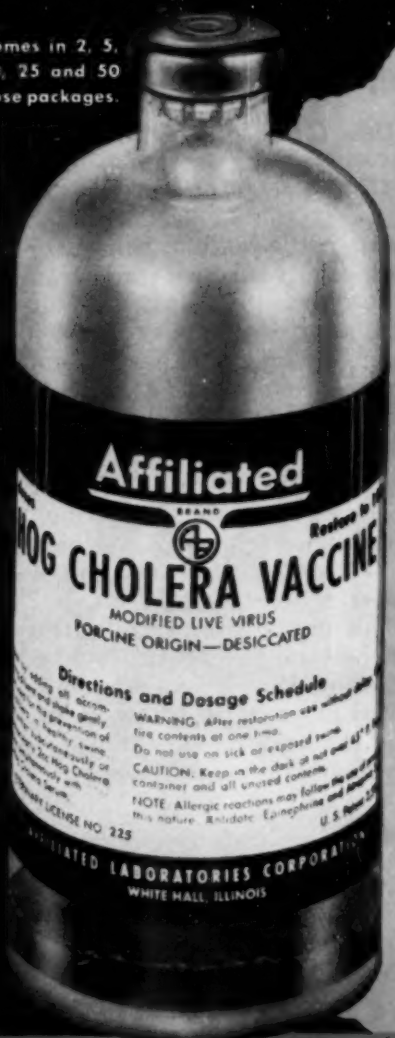
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JOURNAL
of the American Veterinary Medical Association

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Some Factors Responsible for Variations in Viral Immunity

L. E. HANSON, D.V.M., M.S.

Urbana, Illinois

"MEDICINE is most effective in prevention and is least effective in the cure of epidemic diseases. Even today the best medical treatment would not be able to stop any of the pestilences once they got started in a modern American city, by merely caring for the sick."[9]

One important area of preventive medicine is vaccination, a method whereby we try to produce a protective response (antibodies) in a host without serious effect upon the host. We, as a nation, support extensive vaccination programs. This philosophy has measured value but, in addition, it has limitations that are not so well understood. Sometimes the margin between an adequate immunizing dose and a disease-producing dose is very narrow. So, with vaccination, problems arise, stemming in part from the process of antibody production.

VACCINE DEVELOPMENT

Vaccination is not new. A form of vaccination, inoculation, was performed by the people of the Far East centuries ago.[10] Scales and pus from sores of persons with smallpox were transferred to the nostrils of healthy people. Parties were even organized for this purpose. Needless to say, this procedure had some serious drawbacks, e.g., it could introduce the virus into new areas thus precipitating epidemics.

Jenner[10] observed that milkmaids who had developed sores from cowpox were not affected during smallpox epidemics. In 1796, he introduced some material from a lesion on the hand of a milkmaid into the skin of a child. This child later resisted inoculation with smallpox virus. Thus, it was found that a mild virus could produce protection against a more severe, closely related virus.

Pasteur, in his work with fowl cholera, accidentally learned how to reduce the virulence of a bacterium by maintaining the organism in an unfavorable environment. He then applied this knowledge to rabies virus. He placed rabies-infected spinal cords in jars containing potassium hydroxide, thereby drying the tissue and modifying the virulence of the virus.[10] In 1885, he vaccinated a boy who had been bitten by a rabid dog—the child survived. This was an important step forward in the field of preventive medicine.

Our present-day vaccines are based primarily on those early discoveries. For example, fowlpox and laryngotracheitis vaccinations are actually inoculation procedures which have the same advantages and limitations as the original variolation. Both of these vaccines are prepared from relatively virulent strains of virus which could cause an epizootic. Since Pasteur's day, many viruses have been modified to provide valuable immunizing agents. This group includes modified hog cholera, canine distemper, and avianized rabies vaccines, and some of the Newcastle disease and infectious bronchitis vaccines. In addition, inactivated or killed viral agents, capable of producing protective antibodies in an inoculated host, have been utilized. Although these agents do not cause disease, they may not produce high levels of immunity.

VIRUS GROWTH IN ANIMAL TISSUES

To help clarify the problems of virus immunity, a brief review of how viruses grow and multiply may be worthwhile. Viruses are strictly parasitic and must rely on living host cells for reproduction. Present knowledge indicates that the virus particles are composed primarily of nucleic acids enclosed in a thin protein membrane.[1] The nucleic acids appear to represent the active genetic component which, upon entering a cell, is able to convert the cell's machinery (enzyme system) into a manufacturing plant for virus production. In other words, although virus particles contain the controlling mechanism, they are

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Presented before the seventy-fifth annual convention of the Illinois State Veterinary Medical Association, Chicago, February, 1957.

dependent upon the metabolism of living cells for their actual reproduction. Healthy cells usually provide the best environment and, therefore, are the most likely sites for virus invasion. Then, when mature virus particles have been produced, they are gradually released through the cell membrane.

FACTORS IN VARIATION OF IMMUNITY

Mechanism of Antibody Protection.—How can we protect tissue cells from attack by virus particles? There appear to be two logical avenues of approach based on present knowledge. If circulating antibodies can neutralize the virus in the blood stream, cellular invasion does not take place.[1] Most of our vaccines stimulate the formation of circulating antibodies. However, there also is evidence that localized tissue immunity may sometimes play an important role. In this instance, viruses seem to attack the cell walls at specific locations called "cell receptors." The mechanism of local tissue immunity is based on the concept that these receptors have been modified by previous exposure to either an inactivated or modified virus, which prevents attack by the lethal virus.

Viral Variation Caused by an Immune Population.—Because virus particles contain genetic systems, they are capable of modification or of mutation, thus forming a different virus which may or may not be susceptible to antibodies produced in response to the original agent. These changes are presumed to occur because the host is immune to the original virus. Influenza virus of man is perhaps the best-known example of genetic modification or mutation.[1] The influenza virus particles which have resisted the antibodies continue to propagate and produce a new generation of resistant virus particles. The situation is somewhat comparable to the development of DDT-resistant flies. A similar situation which may have been responsible for the appearance of the atypical forms of hog cholera virus has been accomplished experimentally.[3] The influence of hereditary factors may help explain the origin of the different antigenic strains of viruses that occur in equine encephalomyelitis and foot-and-mouth disease.

Mixed or concurrent viral infections comprise an interesting study. We may assume that an animal exposed to two different viruses, such as canine distemper and canine hepatitis, might develop a more severe tissue reaction to both entities. This occurs in the case of these two viruses, and is known as "viral exaltation."[6] A similar pattern has been revealed with herpes virus which has been shown to enhance the susceptibility of mice to pseudorabies virus.[14] However, it is also possible for two different agents to grow in the same host cells, as demonstrated with fowlpox and laryngotracheitis in poultry, with the development of both types of inclusion bodies, yet without enhancing the virulence of either virus.

Viral Interference.—There are other possibilities which may occur as the result of dual or mixed viral infections. Some host cells will not support certain combinations of viruses—a condition known as "viral interference."[12] Newcastle disease virus may be prevented from multiplying under certain conditions by the presence of infectious bronchitis virus in the same host.[11] A reduction in mortality has been demonstrated in chickens which were exposed simultaneously to the two viruses as compared with susceptible birds exposed to Newcastle disease virus alone. No mortality occurred in the chickens given both Newcastle disease virus and infectious bronchitis virus, while a 20 per cent mortality occurred in the birds which received the same concentration of Newcastle disease virus alone.

Viral interference is difficult to demonstrate because many variables are involved. Usually, either the virus in highest concentration, or the one given first, has the advantage. Sometimes this action is reciprocal, at other times not. It seems reasonable to suppose that viral-interfering actions occur in nature. Latent viruses (such as herpes virus of man) or subclinical infections (such as pseudorabies virus in swine) could be responsible for interfering actions. The exact mechanism involved in the interference phenomena has not been established; however, present evidence suggests that the first virus invading a cell utilizes the host cell enzyme system, thus depriving the second virus of a method of metabolism and, therefore, preventing its optimum reproduction.[1]

Interference can also be produced by modified virus or inactivated virus.[8] Resistance to virulent virus has been demonstrated to develop in three days in swine given modified hog cholera virus. The pre-

sumed therapeutic action of Green's distemperoid vaccine when given to a dog previously exposed to virulent distemper has been attributed to the same mechanism.

Genetic Recombination.—Still another possible action, involving viral or vaccinal response, may result when an animal is exposed to two viruses. If the two agents bear a close relationship, as in the case of certain strains of eastern and western equine encephalomyelitis viruses, a crossing-over or recombination of the viral genes may result, with formation of a new genetic strain. This effect has been demonstrated between strains of influenza virus of man and is called "genetic recombination."[1]

Viral Sensitization.—Recently, we have become interested in a phenomenon known as "virus sensitization." In our studies with mixed infections of Newcastle disease and infectious bronchitis viruses, we observed that some birds which were given both agents and failed to show clinical signs had an increased susceptibility upon rechallenge. In one experiment, the chickens rechallenged with Newcastle disease virus sustained a 70 per cent mortality in contrast to a 33 per cent mortality in the control group.

In studies with foot-and-mouth disease, when a small dose of the virus, which failed to produce clinical signs, was followed two weeks later by a more concentrated dose of the virus, greater losses occurred than in animals exposed for the first time.[17] Also, Rocky Mountain spotted fever and epidemic typhus vaccines which lacked adequate antigenicity to produce immunity in animals were reported to increase susceptibility in these animals.[2] A similar observation was made in studies of hog cholera.[4] When swine were given weak hog cholera vaccine, some of them were more susceptible upon rechallenge.

This phenomenon warrants more investigation since it may help explain some of the irregularities following vaccination.

Interfering Action of Congenital Immunity.—Antibodies transmitted from the hen to the chick through the yolk, or from the dam to the calf or pig through the colostrum, have been shown to interfere with development of acquired active immunity.[5,15] In poultry, offspring of immune parents may be immunized with B$_1$ Newcastle disease vaccine administered intranasally at 1 day of age, but these birds are more refractory when given vaccine by other routes. Apparently the intranasal route of vaccination produces a local tissue immunity in the nasal area, which is the natural route of infection. Circulating antibodies give only slight protection, against the virus, to the cells of the nasal mucosa as compared with the protection given to cells below the surface. When the virus is injected parenterally, it may be completely or partially inactivated while being transported by the blood to susceptible tissues. The same congenital immunity mechanism occurs in mammals; therefore, vaccination is best delayed until the young animals are devoid of transient congenital antibodies.

Deficiency of Gamma Globulin.—An interesting, abnormal response to natural or vaccinal infection may occur if the host is incapable of producing normal globulin; the effect being a continued susceptibility due to the failure to develop resistance to an infectious agent. This condition has been found in a few children who have had repeated attacks of measles, scarlet fever, and other bacterial infections. One patient possessed so little *gamma* globulin that he was able indefinitely to support skin grafts from other people.[7] Normally, the antibody response to the foreign protein prevents this except when the graft is from an identical twin.

What can be done to avoid these inconsistencies in immunogenic responses? Some things have already been done and new measures are planned.

Preservation of Commercial Seed Viruses.—The conscientious manufacturer of vaccines is aware of certain pitfalls in the production of virus vaccines and has taken steps to avoid these errors. Because of the occurrence of virus variations and mutations, constant attention is required in the care of seed virus. Each passage of the virus increases the number of generations and the probability of changes in the virus. In an effort to prevent such changes, the number of passages of the seed virus is decreased by lyophilizing and storing large lots of the virus, initially and at occasional intervals, for future use in vaccine production. Thus, the number of generations of the seed virus is held to a minimum.

Improved tests for potency, protection, and sterility are applied to vaccines, but

new and more reliable tests are needed. Such tests can be perfected only by careful research on the part of many workers. Considerable attention has been focused on this problem.[13] These efforts include a joint cooperation between vaccine manufacturers and veterinary pathologists of commercial, educational, and governmental agencies.

SUMMARY

Host response to a viral vaccine is affected by various factors. The genetic construction of a virus may be varied by the immune status of an animal population or through mixed infections in animal cells (genetic recombination). Mixed infections also can result in either exaltation of one or both viral agents or in interference with one or both. Under some conditions, the administration of virus containing inadequate antigen may result in a sensitizing action. Congenital immunity or a deficiency of *gamma* globulin can prevent the development of an adequate antibody response in the host.

A renewed effort is being made to develop a better understanding of both the virus-host relationship and new vaccine-evaluation procedures.

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A New Theory on Virus Activity.—It is reported that the danger factor in a virus is a small element that can emerge only through a tiny opening in the covering of the individual virus particle. Thus it may be possible to alter viruses to render them harmless. This could mean a radical change in methods of immunization. It is possibe that the periodical use of diathermy machines which create artificial fevers could produce immunity to diseases caused by viruses. Some cells have been found to be immune to viruses while others are susceptible.—*Sci. News Letter (April 27, 1957): 265.*

Immunity to Virus Pneumonia in Recovered Pigs.—Following recovery from experimental enzootic virus pneumonia, pigs were strongly resistant to a second exposure. This was not due to increased age since unexposed animals of the same age were as susceptible as younger pigs and all developed pneumonia. Under favorable hygienic conditions, most of the sick animals recovered.—*Nord. Vet.-med. (Feb., 1957): 91.*

Percutaneous Vaccination with a Spray Gun.—Army recruits may soon be vaccinated with an automatic jet injection syringe powered by a small hydraulic pump which produces a jet velocity of 700 ft. per second. The advantages are a saving in time, syringes, and needles. No sterilization is required since the injector nozzle

does not actually touch the skin. Although not completely painless, it causes less discomfort than a needle.—*Sci. News Letter (April 20, 1957): 245.*

An Aid in Making Better Wright's Blood Stains

S. J. SCHILLING, D.V.M., M.S.

Little Rock, Arkansas

Precipitates ruin many slides stained with Wright's stain. This stain, as used, is dissolved in absolute methyl alcohol which is highly volatile. Precipitation is due, usually, to momentary drying of the film during application of the stain and before addition of a buffer solution (or distilled water) to the preparation. The following modification of technique eliminates this problem and assures a higher percentage of good preparations.

The suggested modification requires no change in usual technique but adds one step. Pieces of filter paper slightly narrower than the slide and about 1 inch long (2 by 5 cm.) are cut and kept available. The air-dried film is covered with a piece of this paper and the stain is applied. The paper filters the stain as it penetrates. This eliminates the need for filtration of the stain before application.

As required, stain is added to keep the paper wet and covered. This compensates for evaporation and prevents run-off of stain due to surface tension or other factors. It also prevents the blood film from becoming dry at any time and, thus, avoids precipitation of stain.

After approximately one minute, leaving the filter paper in place, the buffer solution (or distilled water) is applied in usual amounts and left for one to three minutes. The paper offers no impediment to completion of the staining process. The slide is then rinsed (removing the paper), dried, and examined.

In laboratories where many Wright's stain preparations are made and staining jars are used, the outlined procedure would have no advantage. However, in laboratories where individual slides are prepared by direct application of the stain, this modification is helpful. This method is of greatest advantage in hot, dry weather.

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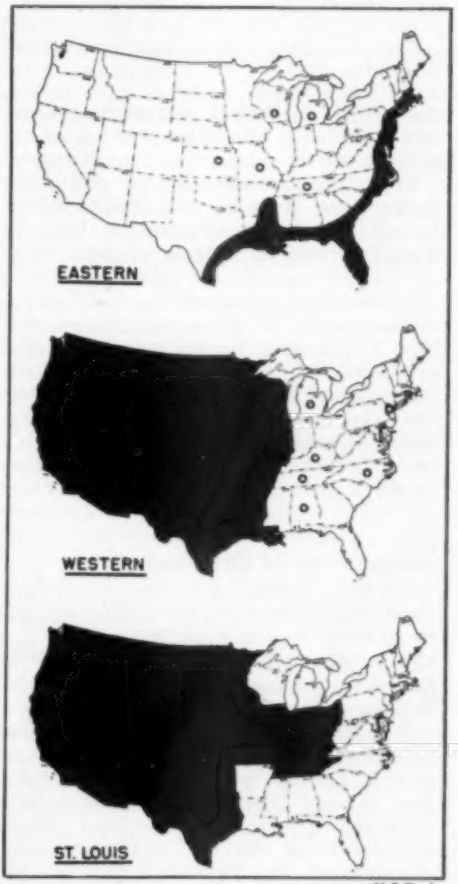
Equine Encephalomyelitis in 1956

Encephalomyelitis was reported in 1,284 equine animals in the United States in 1956, slightly more than in the two previous years but, with two exceptions (1950 and 1951), less than in the previous 19 years. In 1937, there were 173,889 cases; in 1938, 184,662 cases. The 493 deaths were the lowest in 22 years except in 1951 and 1954. Of the ten states in which the infection was identified as the eastern type of virus, 270 (87%) of the 310 infected animals died. Of 303 cases of encephalitis in man, most were caused by the St. Louis



—U.S.D.A.

Fig. 1—Geographical distribution of the arthropod-borne encephalitides in the United States (total virus activity in man and animals) through January, 1957. The blackened areas show repeated history of virus activity within the state; circles indicate history of isolated occurrence within the state.

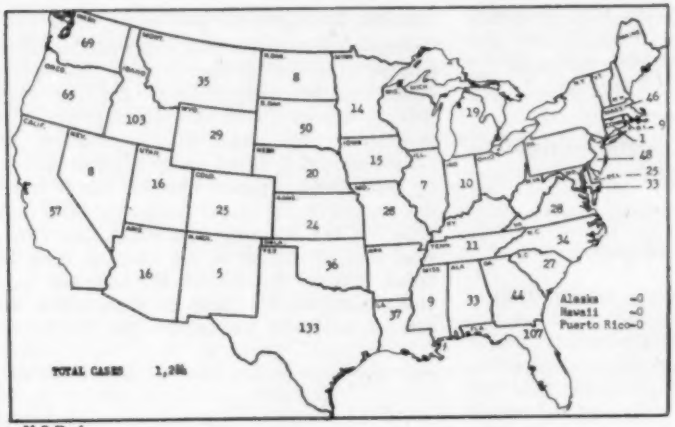


Fig. 2—Equine encephalomyelitis reported during the calendar year 1956.

—*U.S.D.A.*

encephalitis virus but eastern-type virus was identified in 12 and western equine encephalomyelitis virus in at least 39 cases.

In addition to pheasants (800 died of the eastern-type virus on two Rhode Island farms) and a few chickens, at least 30 species of wild birds were affected.

Equine Encephalomyelitis in Syria.—Encephalitis of horses and donkeys has been investigated for six years in Egypt and Syria, and has been produced experimentally in chickens, kittens, and sheep. The disease occurs in horses in early spring and subsides by June. In Syria, it is probably of greater economical importance in sheep than in horses.—*Nature (March 16, 1957): 584.*

Transmission of Ornithosis

Ornithosis virus was not recovered from 1,532 dead embryos or 630 poults obtained from 2,954 eggs laid by 165 breeder turkeys while they were in the viremic or postrecovery stages of an experimental infection.

When fresh turkey eggs were inoculated with ornithosis virus, the agent was not recovered beyond the ninth day of incubation. When 7-day-old turkey embryos were inoculated, the mortality was high and ornithosis virus was not recovered beyond the sixteenth day. Ornithosis virus was not recovered from eggs laid by hens in the viremic stage of ornithosis when isolation attempts were made on freshly laid and 24-hour incubated eggs. Ornithosis virus lived no longer than three days on turkey eggshells or four days on cotton swabs under egg-incubation conditions.—[*D. E. Davis, J. P. Delplane, and J. R. Watkins: The Role of Turkey Eggs in the Transmission of Ornithosis. Am. J. Vet. Res., 18, (April, 1957): 409-413.*]

Avianized Virus Vaccine for Canine Distemper.—Using Green's distemperoid virus passaged through embryonating eggs, contagiousness for ferrets disappeared at the twenty-fifth passage, and attenuation was adequate at 130 passages for immunizing dogs and ferrets. Using 500 egg-infective doses per dog, approximately 40,000 were satisfactorily immunized in field tests with this Onderstepoort strain vaccine.—*D. A. Haig in Onderstepoort J. Vet. Res., 27, (1956): 19-53.*

Infectious Bronchitis Vaccine.—A satisfactory immunity for ten months resulted when chickens of different ages were exposed to an infectious bronchitis virus produced by 42 to 67 passages through embryonating chicken eggs.—*M. S. Hofstad in Vet. Med. (Oct., 1956): 464.*

Immunity to Taenia Hydatigena in Sheep.—An absolute immunity was acquired against *Taenia hydatigena* by 2 lambs initially infected at pasture. A similar resistance was demonstrated in 10 lambs seven weeks after they had been given 50 to 800 ova.—*Canad. J. Comp. Med. and Vet. Sci. (March, 1957): 65.*

Naturally Occurring Infection with Equine Encephalomyelitis Virus in Turkeys

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SINCE 1938, numerous reports have been made of natural eastern equine encephalomyelitis virus infection in pheasants. It has been reported in Connecticut[1] and in New Jersey.[2,3]

Western-type virus was recovered from a prairie chicken in North Dakota[4] and from a nestling magpie and nestling red-winged black birds in Colorado.[5] Many additional instances have been recorded of naturally occuring eastern and western types in various species of animals and birds.

Animals and birds of many species, both wild and domestic, may develop infection through intracerebral and, in some instances, subcutaneous or intramuscular inoculation of the virus.

Mosquitoes, ticks and mites, and lice of wild and domesticated birds are capable of transmitting the virus. Several genera of mosquitoes are incriminated as the chief vectors of the virus from birds which, possibly, are important reservoirs of encephalomyelitis virus.

Two turkeys showing nervous signs were brought to the diagnostic laboratory on Sept. 4, 1956, from a flock of 10,000 hens and toms of market age, located near Lincoln, Neb. One turkey was living, the other had died enroute. The owner reported a loss of 15 to 20 birds per day during the preceding three or four days. Approximately 100 were sick and off feed.

The history indicated that the dead and ailing turkeys were about equally divided between toms and hens. Many were found dead; however, the sequence of clinical signs consisted first of drowsiness, ruffled plumage, tremors, staggering gait, and refusal to eat or drink, then progressive weakness and complete leg paralysis. The affected birds rested on the breast with head either drawn back or neck extended with head resting on the ground. In acute cases, death occurred in two to three days.

The live turkey showed typical signs of the advanced stage of the disease as described. Food poisoning was considered, but the only recent change was the addition of newly harvested milo to the ration. However, this was excluded as being a factor since another flock of 3,000 birds on the same ration and maintained separately under like conditions was unaffected.

At necropsy, the entire digestive tracts were empty and, aside from distended gallbladders and a slight duodenitis, no gross pathological changes were found. Bacteriological examination for usual pathogens proved negative as did the hemagglutination-inhibition test for Newcastle disease. No therapy was recommended but the owner was advised to separate the ailing birds from those apparently healthy.

On September 17, 7 more turkeys were brought to the laboratory showing signs of various stages of the disease. The owner stated that losses had averaged 15 per day during the previous two weeks but the number of new cases seemed to be declining. Many of the separated sick birds had died. Some had been affected two weeks or longer and a few had apparently recovered. Rapid dehydration and emaciation were observed in those ill over a prolonged period.

Equine encephalomyelitis infection was suspected, due to the similarity to the disease in pheasants. No known cases in animals had been reported from this area. Mosquitoes had been unusually numerous in the vicinity during late summer and fall.

Blood was drawn from 3 birds, and the brains, spleens, and heart muscle aseptically removed. Serum was separated from a portion of the blood, followed by quick freezing of the specimens. With the exception of a slight air sac infection in 2 of the 7 turkeys, gross lesions were not found. Serological (Newcastle disease) and bacteriological examinations of the specimens again were negative.

By the time the owner and state officials were advised of the tentative diagnosis, the apparently well birds had been sold to a processing plant. Those obviously affected

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were withheld from the market and later destroyed.

Frozen turkey brains, serum, and whole blood packed in dry ice were sent by air express to the Communicable Disease Center (C.D.C.) of the U. S. Public Health Service, Montgomery, Ala., following communication with the Kansas City, Kan., field station.

The initial report from C.D.C. dated Oct. 3, 1956, stated that virus was not isolated through mouse inoculation. This was probably due to the transient nature of the virus. It has been reported[6] that virus was not found to persist in any organs of the chicken for more than three days after inoculation and in most instances did not persist over two days. Antibodies, however, were present in the blood within 15 days postinoculation.

The subsequent and final report from C.D.C., dated October 10, stated that the neutralization index of the turkey serum against western equine encephalomyelitis virus was in excess of 500.

As far as the writer can determine, this is the first published report of a natural infection of turkeys with equine encephalomyelitis virus.

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Allergenicity of Tranquilizing Drugs.—Meprobamate, an effective tranquilizer with low toxicity, is reported, from Florida, as giving rise to allergic reactions in 15 patients. The reactions include urticaria, temperature of 40 C., arthralgia, and purpura. The previous use of mephenesin may have presensitized some of these patients. Reserpine and chlorpromazine have caused side effects of a more variable type. Percentagewise these allergic reactions are low but they must be borne in mind since they may cause confusion in making a diagnosis.—*J.Am.M.A. (March 16, 1957): 930.*

Drug Fever.—Pyrexia caused by idiosyncrasy to a drug is reported in 53 patients, 25 had been given a sulfonamide, 4 penicillin. In 34 cases, the patient was normal except for the fever; the other 19 showed additional changes such as a rash. In 13, the pulse rate did not rise with the temperature, suggesting that this was an allergic reaction.—*J.Am.M.A. (Feb. 16, 1957): 571.*

A Report on *Fungal Diseases of Man.*—Records on 15,845 patients at a hospital in St. Louis, over a 35-year period, revealed 88 patients with fungal infections. Of the 88, there were 20 cases of histoplasmosis, 13 of cryptococcosis, 9 of mucormycosis, 25 of candidiasis, 3 of aspergillosis, 9 of actinomycosis, 7 of blastomycosis, and 2 of coccidiomycosis. The infection was primary in 41 patients and secondary in 47.

While the incidence of primary fungal infections did no change when antibiotics came into use, a striking increase occurred in the secondary infections, especially histoplasmosis, cryptococcosis, mucormycosis, candidiasis, and aspergillosis. Of the 47 secondary fungal infections, 40 occurred after 1942 and 30 in persons treated with antibiotics for at least seven days.—*J.Am.M.A. (March 9, 1957): 881.*

Mucormycosis in Man.—This new disease is reported with increasing frequency, probably due to the use of antibiotics which, by reducing bacteria, permit fungus invasion. Cortisone and antileukemic drugs may also favor the development of this mycosis. The usual cause is Rhizopus, a ubiquitous laboratory contaminant which enters the nose and leads to sinusitis, orbital cellulitis, meningoencephalitis, or to pulmonary invasion. It may be fatal in one day to one month.—*J.Am.M.A. (March 9, 1957): 805.*

Fungistatic Agents.—Resorcinol monobenzoate was the most fungistatic of several resorcinol derivatives tested. It was

effective at 0.1 per cent and 0.01 per cent, did not produce conjunctival irritation, and was one half as toxic as the clinically used resorcinol monoacetate.—*J. Am. Pharm. A. (March, 1957): 185.*

Newcastle Disease in Man.—A man developed a virus infection of the lungs after entering a chicken house which had been dusted with Newcastle disease (B-1 strain) and infectious bronchitis vaccine. There is no record of infection in man caused by the virus of infectious bronchitis, but the Newcastle virus has caused acute granular conjunctivitis of short duration in some persons. Lower respiratory tract involvement has been uncommon in this disease. Serological tests were not reported.—*J.Am. M.A., (March 9, 1957): 909.*

Chronic Brucellosis in Man.—In man, both acute brucellosis and chronic localized brucellosis are accepted clinical entities but disagreement exists concerning generalized "chronic brucellosis." One case of 14 years' duration, the diagnosis substantiated by cultural methods, is reported in a man with severe splenomegaly as the chief sign. The duration, continued constitutional reaction, degree of splenomegaly, and the thrombocytopenia are unusual features of this case.—*Proc. Mayo Clinic (Feb. 6, 1957): 58.*

At a recent meeting in Ohio, 178 veterinarians were tested serologically for leptospirosis, psittacosis, brucellosis, and Q fever. Of these, 32 had titers against only one of these diseases, 5 were positive for 2 antigens. The highest incidence of infection was found in the 88 large animal and general practitioners. Of these, 12 were positive for Q fever, 10 for brucellosis, 4 for psittacosis, and 1 for leptospirosis.—*Anim. Dis. Trends, Jan., 1957.*

With the world demand for laboratory animals increasing enormously, an International Commitee on Laboratory Animals has been established to insure an adequate supply, to improve the quality, and to standardize terms used with respect to such items as genetics, diseases due to parasitism, nutrition, care, and performance records of these animals.—*Science (April 19, 1957): 753.*

Leptospirosis and Brucellosis Titers in Deer Blood

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The role of wildlife in the spread of disease is an interesting phase of epizootiology. The deer population in Minnesota, during 1956, was estimated at 400,000. Considering this population and the fact that a common area of pasturage and source of water often exists between cattle and deer, it was believed that a wildlife reservoir of leptospirosis and brucellosis might exist in the deer of Minnesota. An investigation was undertaken to obtain serological evidence of this.

METHODS AND RESULTS

With the assistance of various sportsmen's groups, game biologists, and veterinarians, approximately 400 samples of deer blood were received, 187 of which were considered satisfactory for study. The remainder were either badly contaminated or hemolyzed. Information as to age, sex, and location of kill was lacking on many samples but sufficient data were obtained to indicate that a broad coverage of several factors was represented.

TABLE I—Serological Findings of Leptospirosis (Leptospira Pomona Antigen)

No. of samples	Titers	Per cent
157	Negative	84.1
8	1:10	4.2
14*	1:100	7.5
4	1:1,000	2.1
4	1:10,000	2.1

*Cross reactions in two samples with Leptospira icterohemorrhagiae antigen at 1:10 dilutions.

The 187 satisfactory samples were tested in the laboratory for leptospirosis by the agglutination-lysis test, using live *Leptospira pomona* and *Leptospira icterohemorrhagiae* antigens. The blood serum-agglu-

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The authors thank Dr. Arnold Erickson, Minnesota Game and Fish Division, Bureau of Research and Planning; Dr. Ralph L. West, executive officer, Minnesota State Live Stock Sanitary Board; Dr. Jay Sautter, head Division of Pathology and Parasitology, School of Veterinary Medicine, University of Minnesota, for helping to make this investigation possible.

tination plate test was used for determining evidence of Brucella infection.

The results are shown in tables 1 and 2.

TABLE 2—Serological Findings of Brucellosis (Rapid Plate Test)

No. of samples	Titers	Per cent
178	Negative	99.45
1	1:100	0.55

Only 179 of the 187 samples were tested for brucellosis due to excessive hemolysis.

CONCLUSIONS AND SUMMARY

Serological titers to *Leptospira pomona* were found in Minnesota deer. Of the samples tested for leptospirosis, 157 (84%) were negative, while 30 (16%) showed some degree of reaction. Two samples showed cross reactions to *Leptospira icterohemorrhagiae* antigen at 1:10 dilution. Animals found positive to the leptospirosis test varied as to age and sex, indicating no apparent influence from these factors. The varied locations in which reacting deer were killed indicates that this disease may exist in several areas in Minnesota.

Of the 179 samples tested by the rapid plate test for brucellosis, only one was positive. Since the titer of the reacting sample was relatively low, it appears doubtful if active Brucella infection existed in any of the deer represented.

Biological Effects of Radiation.—Experiments show that every irradiation of an animal body results in some irreparable damage and that the lethal dose gradually decreases with the age of the animal. Irradiation apparently accelerates the natural processes of aging and thus shortens the life span. Also, quite apart from genetic effects, fetuses of human as well as animal species can be damaged by large doses of radiation to the mother's abdomen. Such injuries as arrested development of the central nervous system may occur.—*Nature (April 13, 1957): 755.*

Radioactive Fall-Out Failed to Cause Cancer.—Cattle accidentally exposed during an atomic explosion in New Mexico, in 1945, have shown a graying and loss of hair, extensive crusts and prominent overgrowth on the skin of the back, but no signs of cancer 12 years later. Human inhabitants of Marshall Island, exposed in March, 1954, also show no serious consequences three years later.—*Sci. News Letter (April 20, 1957): 248.*

Cerebral Ataxia in a Man Due to DDT and Lindane Poisoning.—A butcher exposed daily to vapors of DDT and lindane in his "kill room" developed a neurological condition typical of cerebral ataxia and neuronitis resembling that in animals chronically exposed to DDT. He gradually recovered without treatment when hospitalized.—*Proc. Mayo Clinic (Feb. 20, 1957): 67.*

Careless use of pesticides causes the death of 100 to 150 persons each year. Many are children who have played with the pesticide containers.—*Sci. News Letter (April 20, 1957): 253.*

Porcine Atrophic Rhinitis in Romania.—Atrophic rhinitis in swine, first detected in Romania in 1954, was studied in nine herds. Clinical signs were found in 3.5 per cent of the 13,118 swine (348 pigs and 119 adults). The incidence of identified organisms isolated from the nasal discharge included: *Pseudomonas pyocyanea* (47.3%), *Bacterium coli* (13.4%), *Fusiformis necrophorus* (7.3%), Pasteurella (7.2%), *Corynebacterium pyogenes* (4.5%), and Trichomonas (35.2%). Pigs 14 days old remained healthy when inoculated with cultures of these organisms, but when inoculated with a suspension of nasal discharge, pigs 2 to 5 days old developed the disease. —*Vet. Bull., Item 557 (Feb., 1957): 87.*

Erysipelothrix rhusiopathiae, with cultural characteristics resembling those isolated from cardiac vegetations in swine, were isolated from the tonsils of 9 of 240 apparently healthy cattle in Romania. Cultures killed mice in five to nine days and pigeons in four to eight days.—*Vet. Bull. (March, 1957): 106.*

Guinea Pigs Immunized Against Ascaris. —Immunity comparable to that produced by normal infection against *Ascaris lumbricoides* can be induced by subcutaneous injection of embryonating and infective ova, creating a depot of viable eggs from which larvae wander locally in the tissues; they may reach the lungs.—*Nature (April 13, 1957): 783.*

Surgery and Obstetrics
and Problems of Breeding

Preliminary Report on Repair of Coxofemoral Luxation and Coxofemoral Subluxation in Cattle

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COXOFEMORAL luxations and subluxations in cattle have long been a problem to the practicing veterinarian. Many cows have been slaughtered or otherwise have met an untimely end because of their vulnerability to these conditions, largely due to the shallow acetabulum characteristic of the species. Some of the many cases brought to this clinic have developed a "false joint" with the head of the femur out of the acetabulum. Persistent lameness, reinjury from breeding, and abuse from other cattle often prevent a cow from coming back to good milk flow or from becoming a useful breeding animal.

INCIDENCE AND OCCURRENCE

Although not a large number of animals are affected annually with coxofemoral luxation or subluxation, each case constitutes a chronic lameness and, in most cases, an animal that is lost for breeding purposes or economical milk production. More cases occur in cows than in bulls. However, a bull affected with either condition is of no use except for possible artificial collection of semen with an electric ejaculator.

A high percentage of the cases in cows occur following calving. Incoordination, produced by a partial or complete obturator nerve paralysis, or milk fever, plus the relaxation of the sacrosciatic ligaments can predispose to luxation. Other cases have the history of lameness following a heat period, probably due to slipping and falling while being mounted by a bull or by other cows. Falling from any cause, such as high doorsills in barns, slipping on ice, and loading injuries, account for the rest of the cases in our clinic.

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Presented before the combined sections on General Practice and Surgery and Obstetrics, Ninety-Third Annual Meeting, American Veterinary Medical Association, San Antonio, Texas, Oct. 15-18, 1956.

The author thanks Dr. Jack E. Catlin, Montana State College, for his valuable help in developing the pin and guide apparatus used in this problem; and the members of the Department of Anatomy, Colorado A. & M. College, who aided in overcoming the anatomical problems involved.

CLINICAL DESCRIPTION

In many cases of complete luxation of the coxofemoral joint, the head of the femur rides above the acetabulum, making the leg appear to be somewhat shorter, and the toe of the affected limb turns inward. In other cases, the head of the femur may be in the obturator foramen, causing a distortion of the area over the hip joint. In either situation, both a supporting and swinging leg lameness is exhibited. The animal first shows severe pain, and some individuals will refuse to rise. Cows that are in advanced pregnancy have extreme difficulty in rising and sometimes never get up. A large swelling usually develops over the coxofemoral joint and the femur can often be forced to change positions. The lameness decreases after several days and, eventually, a false joint forms if the animal does not become moribund. Once a false joint forms, a distinct interference with locomotion remains with the animal. Crepitation continues to be noticeable in some animals.

In subluxation of the coxofemoral joint, the round ligament is torn but the joint is not completely luxated. In this clinic, this condition is seen more commonly than complete coxofemoral luxation. Swelling and crepitation over the joint are prominent signs of coxofemoral subluxation. The animal will show considerable pain and lameness. If the head of the femur is seated in the ventral notches of the acetabulum, a loud popping sound will ensue when it slips into place as the animal walks. The toe may point inward, in some cases, without the joint being completely luxated. The lameness will gradually decrease over a period of three to four months but some lameness will persist and the joint is subject to reinjury at any time. There is apparently no regeneration of the round ligament.

DIAGNOSIS

X-ray and rectal examinations are helpful in diagnosing complete luxations. If the head of the femur is still in the ace-

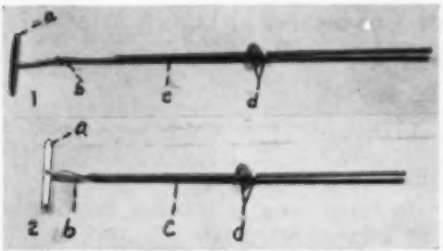

Fig. 1—Original shuttle-pin apparatus; (a) Leighton shuttle pin, (b) 18-gauge stainless steel wire, (c) threaded 3/16-inch stainless steel shaft, and (d) stainless steel washer and nut.

Fig. 2—Modified shuttle pin apparatus; (a) stainless steel shuttle pin, (b) stainless steel airplane cable, (c) stainless steel 3/16-inch threaded shaft, and (d) stainless steel washer and nut.

tabulum but the round ligament is torn, rectal examination and external palpation are used while the animal is walking. The swelling over the coxofemoral joint is also an adjunct to diagnosis in both types of cases. In complete coxofemoral luxation, the displaced femur can be palpated, in most cases. Pelvic fractures may complicate coxofemoral luxation or may occur independently and should be ruled out by rectal examination. The animal may refuse to rise, making complete examination difficult.

Coxofemoral luxation may complicate milk fever, and all "downer cows" should be examined for this condition. At times, it will be difficult to make a differential diagnosis of coxofemoral luxation from fractures of the pelvis or femur without rectal palpation and a radiograph. A small cassette may be inserted into the rectum to obtain a radiograph of the coxofemoral junction[4] which will show whether there is complete luxation or just a separation of the joint which indicates a torn round ligament. Stifle injuries, such as traumatic rupture of the collateral ligaments of the stifle joint, may produce crepitation and must be differentiated by careful examination.

Fig. 3—Lateral view of the proximal extremity of the right bovine femur. Arrow points to spot for drilling.

THERAPY

Previous treatment has consisted of placing the animal in confinement in a small stall, making feed and water easily accessible, and keeping exercise at a minimum. In many cases, the luxation was reduced by manipulation, with the animal anesthetized, only to have the condition recur in a short time.

When a shuttle pin method for correcting coxofemoral luxation in the dog was described,[3] it was decided to attempt a similar repair in cattle. Since it was obvious that a fascial or plastic suture material was not strong enough to replace the round ligament in cattle, an artificial ligament was prepared from 18-gauge stainless steel wire, a stainless steel shuttle pin,* and a 3/16-inch stainless steel threaded shaft (fig. 1). Double or triple strands of the wire, forming a cable approximately 1½ inches long, were attached to the shuttle pin and to the stainless steel shaft to furnish some flexibility where the acetabulum meets the head of the femur. This material was not strong enough for all cases so the wire was replaced with stainless steel airplane cable (fig. 2) with a dead weight breaking point of 800 lb., compared to 165 lb. when made with 18-gauge stainless steel wire. This should hold all the stresses of any size animal at the coxofemoral junction.

The corrective apparatus must be arranged so that the shuttle pin can be turned after it enters the pelvic canal (fig. 7). To insure flexibility at the junction of the femur and acetabulum, the nonrigid portion of this artificial round

*Leighton shuttle pin.

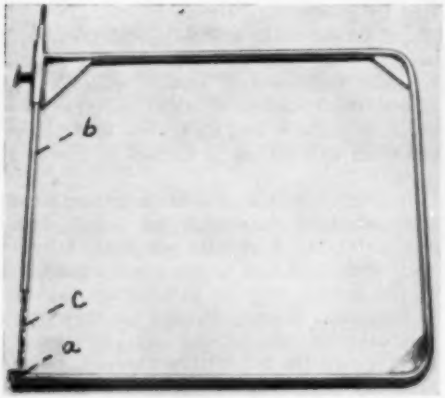

Fig. 4—Guide apparatus for drill; (a) guide spot for centering drill, (b) guide for drill, and (c) drill—showing final position.

ligament should extend into the head of the femur approximately 1 inch. Stainless steel washers and nuts must be used so that electrolytic action of the metal can be kept at a minimum.

THE OPERATION

An area 12 by 18 inches, with the center over the great trochanter of the femur, is clipped and shaved. A 2 per cent procaine solution or other suitable anesthetic is given epidurally in sufficient quantity to weaken the hind parts, so the animal can be gently cast with the aid of a rope. After the epidural anesthetic is fully effective, chloral hydrate (7%) solution is given intravenously until the animal is in deep narcosis. The shaved area is then scrubbed with soap and water and suitable skin antiseptics are applied. The area of the incision is then infiltrated with 2 per cent procaine to minimize the general anesthetic needed. The operative site is draped and an incision is made lateral to the femur, over the greater trochanter. The incision is started approximately 3 inches above the proximal end of the trochanter major and extends downward parallel to the femur for 8 inches. A bony eminence, which is part of the area of insertion of the gluteus medius muscle, just below the trochanter major, is used as a landmark for drilling (fig. 3). The fascia and the biceps femoris muscle are incised until the eminence of the femur is exposed. Before drilling, the femoral head must be in the acetabulum. If its position has not been determined by

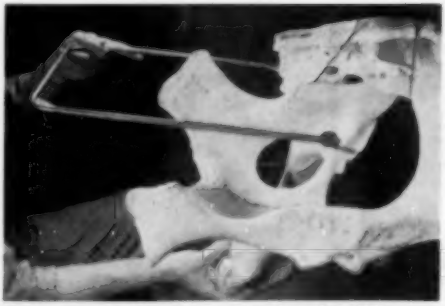

Fig. 5— Guide apparatus in place for drilling.

radiograph, the gluteus medius muscle can be divided above the great trochanter and the coxofemoral junction can be palpated with one finger.

Usually, when completely luxated, the head of the femur is above the acetabulum on the ilium. This is corrected by applying traction to the leg (calf puller or block and tackle) and lifting on the trochanter major by means of a one-finger obstetrical hook. Other luxations are corrected according to their position by applying traction and lifting on the femur to force it back to the acetabulum. Once the head is in place, and the leg is in a normal position, the drilling through the femur and acetabulum can be done.

A small piece of bone is chiseled from the eminence to allow entrance of the ¼-inch drill. One extremity of the guide apparatus (fig. 4) is inserted into the rectum by the assistant, who must be careful not to touch the shaft which will guide the drill through the femur and acetabulum. The round disc on this extremity is held, by the assistant, over the area on the pelvic side of the acetabulum where it is desired to have the drill come through (fig. 5). After proper angulation, drilling is started. When the point of the drill penetrates the acetabular wall, the assistant informs the operator and drilling is stopped. The drill is then removed and the shuttle pin is inserted in a position parallel to the wire and the threaded shaft (fig. 6). The assistant then turns the shuttle pin with the hand which is in the rectum (fig. 7, 8). A stainless steel washer and nut are placed on the threaded shaft and screwed into position until the desired degree of firmness is obtained. The firmness of position of the pin is tested by moving the leg in all direc-

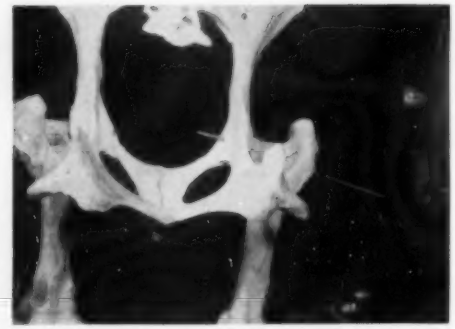

Fig. 6—Shuttle pin in position before being turned.

Fig. 7—Shuttle pin turned at a right angle to the cable.

Fig. 8—Shuttle pin in its final position.

tions, making sure that the animal can obtain the proper degree of abduction without putting too much stress on the pin or on the bone. The excess of the pin shaft is then cut off and the stump is flared to be sure that the nut can not loosen and come off.

First, the gluteus medius muscle, then the biceps femoris, is brought into apposition by means of a continuous suture with No. 2 catgut using a swedged-on traumatic needle. The skin is sutured with a continuous interlocking stitch with No. 2 monofilament nylon or other suitable noncapillary suture, and a surgical dressing of collodion and cotton is applied to the incision.

In young animals, a method similar to the technique developed for dogs,[3] using a stainless steel shuttle pin and stainless steel wire anchored to the great trochanter of the femur, may be satisfactory.

Antibiotic therapy is used for three days following surgery. The animal usually start using its leg within three days and recovery progresses to its maximum in approximately six weeks.

There will sometimes be a permanent lameness, but the cows will be able to function in the breeding herd under pasture conditions.

RESULTS

Seven cattle, including 1 calf, 8 months old, and 1 bull, have recovered satisfactorily with this operation. The bull is presently breeding cows and all of the cows are milking satisfactorily with little interference in their gait. One of these cows also had a complicating fracture of the ilium.

Results were poor in 3 cows. One was euthanized when necrosis of the head of the femur and of the acetabulum were found at the time of surgery. In 2 cows, which had obturator paralysis at the time of surgery, the wire strands broke when the leg slipped out in the typical obturator paralysis position.

One normal cow, operated on experimentally, showed no ill effects from the shuttle pin apparatus when slaughtered 18 months later.

DISCUSSION

Many problems were encountered in developing a proper drilling guide and the shuttle pin apparatus for repairing coxofemoral luxation or subluxation. Even with a guide apparatus, considerable judgment must be used, and the basic anatomy should be reviewed before each operation.

In the operation on the calf, anchoring wire strands were used from the shuttle pin to the greater trochanter of the femur.

Following breakage of the wire strands in the 2 cows with obturator paralysis,

stainless steel airplane cable that will probably never break was substituted for the wire.

Since recovery from coxofemoral luxation or subluxation is extremely rare, it is hoped that this limited work will encourage others to further improve the technique of repair.

SUMMARY

A surgical method for repair of coxofemoral luxations or subluxations, similar to the Knowles operation for dogs, was successfully used in 7 of 10 cattle. The special equipment, apparatus, and procedure used is described.

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Serum Mineral Levels in Parturient Cows.—In normal parturient cows, serum calcium levels were lowest one day after parturition and were significantly lower in older than in younger cows. Serum magnesium was highest one day prior to calving, being highest in older cows. Total phosphorus decreased during 24 hours prepartum and was lowest at parturition, especially in older cows. Plasma phosphate rose in cows after calving, with a secondary fall two days later.—*J. Comp. Path. and Therap., 65, (1955): 20-36.*

Hypoparathyroidism and Parturient Paresis.—Hypoparathyroidism is characterized by hypocalcemia, *hyper*phosphatemia, and a decreased renal elimination of calcium and phosporus, while in parturient paresis there is hypocalcemia, *hypo*phosphatemia, and no change in renal elimination of calcium and phosphorus.—*Ann. New York Acad. Sci., 64, (1956): 386-397.*

Bone Marrow Changes in Parturient Cows.— Bone marrow studies of 15 cows, one to eight days after normal parturition, and of 15 cows, one to ten days after retaining the fetal membranes, showed a neutropenia, a marked reticulocyte reaction, and a marked shift to the left in the neutrophils in the latter group of cows. These changes resembled those in human bone marrow in cases of toxic or anaphylactic agranulocytosis.—*Vet. Bull., Item 618 (Feb., 1957): 96.*

Interesting Items on Cattle Breeding.—A comparison of breeding efficiency in four dairy breeds, over 25 years at Oregon State College, showed the record of the best to be 13.7 per cent better than the poorest. Significant breeding efficiency differences occurred between families within the same breed.

Estrus occurred in 6 per cent of the pregnant cows at Ohio State University, usually two to three months after conception.

Involution of the uterus seemed complete in 6 per cent of Wisconsin cows, 30 days after calving; in 75 per cent after 60 days; in 96 per cent after 90 days; and in 99 per cent after 105 days. The uterus returned to normal an average of eight days earlier in young than in old cows. The conception rate of cows bred at the first postparturient estrus was 35.7 per cent; at the second estrus, 70.6 per cent. The first postparturient estrus in normal cows occurred 35 to 55 days after calving.—*Guernsey Breeders' J. (April 1, 1957): 729.*

Activating Bovine Spermatozoa.—In a field trial with 1,808 cows, when semen which had been stored one or two days was activated with sodium carbonate (0.1%), it resulted in a conception percentage of 71.6 compared with 65.9 for controls.—*J. Dai. Sci. (March, 1957): 203.*

Treating Ovine Pregnancy Toxemia.—Results of treating ewes with pregnancy toxemia, in New Zealand, were all disappointing. The most successful (36 to 50% recovery) therapy was 2 oz. of glycerol *per os* twice daily, given alone or with 40 I.U. of insulin, 40 I.U. of ACTH, or thiamine (100 mg.) and cortisone (150 mg.). When methionine was given twice daily, 25 per cent recovered compared with 20 per cent recovery in controls. Death appeared to be hastened when either glucose or calcium borogluconate solution was given intravenously.—*New Zealand Vet. J. (Dec., 1956): 136-144.*

Meningioma in a Dog

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A male, mixed Terrier, 9 years old, weighing 20 lb., was believed to have been struck by a car on March 11, 1955, and to have suffered a brain concussion. The pupillary reflexes were unequal but neither fractures nor internal injuries were discernible. There was a slight swelling on the right side of the head medial to the right ear.

Three weeks later, the dog again became prostrate but recovered. The swelling was slightly larger.

On Jan. 6, 1956, the dog was again prostrate and had a temperature of 103 F. The swelling on the head was slightly more enlarged and, although it was firm, abscess formation resulting from the injury was suspected. The dog again recovered but, during the next four months, it had two similar episodes of prostration and vomition.

A radiograph of the cranial region (fig. 1) was taken May 3, 1956. The diagnosis, by a neurosurgeon, was a tumor originating within the cranial cavity. Upon operation, the mass proved to be a tumor measuring 4.5 cm. in diameter and extending outward from the meninges through the parietal region. The dog survived only six hours after the operation which required nearly three hours.

Histologically, the tumor was thought to be consistent with a meningioma; a rare tumor in the dog.

The tumor probably was present previous to the initial attack. The contusion may have caused enough trauma to account for the pupillary changes seen at the time.

Drs. Baronti and Rhodes are practitioners in Eugene, Ore.

Hyaluronidase in Local Anesthetics.—Since experiments in 250 animals showed that hyaluronidase accelerated healing of infections caused by streptococci, staphylococci, and viruses, this drug was mixed (6 I.U./ml.) with a local anesthetic for use in treating infections of the hand. It reduced the pain of the injection, soft tissues were not distorted, and landmarks were not obscured. Also, anesthesia was obtained in about half the time, sensation returned quickly, and there was less afterpain. Healing was not delayed.—*J.Am.M.A. (Feb. 16, 1957): 588.*

Fig. 1—Radiograph of lateral view of the head of the dog. Arrows show the area of the tumor.

Congenital Malformations of the Heart and Great Vessels in Dogs—A Report of Three Cases

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ORMAND C. JULIAN, M.D., Ph.D; WILLIAM J. GROVE, M.D.

Chicago, Illinois

CONGENITAL malformations of the heart in dogs have been infrequently identified and reported. During the last 12 months, 2 cases, 1 of patent ductus arteriosus and 1 of tetralogy of Fallot, were found among 250 dogs whose hearts and great vessels were examined in our laboratory. A ductus arteriosus was previously found in a dog used in a physiology teaching laboratory.

Patent ductus arteriosus is mentioned[1] as being the most common congenital heart defect. This ailment was found[2] seven times in 13 cases of heart disease in dogs. The tetralogy of Fallot in dogs has not been described; however, isolated malformations, such as hypertrophy of the right ventricle, pulmonic stenosis, and interventricular septal defects, have been reported.[1-5]

CASE REPORTS

Case 1—Patent Ductus Arteriosus and Aneurysmal Dilatation of Aortic Arch with an Embryonic Septal Remnant.—This mixed breed dog, 2 years old, first seen in April, 1956, showed no physical defects.

Upon entering the chest for experimental cardiac surgery, a dilated aortic arch was noticed. Palpation revealed a systolic thrill over the dilated portion of the aortic arch and a continuous thrill with systolic accentuation over the left pulmonary artery. Both thrills disappeared during manual compression of the aortic arch. The heart was normal in color, shape, and size. A patent ductus arteriosus was identified. Pressures were measured through a 15-gauge needle connected to a mercury manometer. The mean pressure in the dilated portion of the aortic arch was 36 mm. Hg and that in the bifurcation of the pulmonary artery, 14 mm. Hg. The mean pressure in the pulmonary artery dropped from 14 to 8 mm. Hg upon occluding the ductus arteriosus.

At necropsy, the patent ductus arteriosus measured 1.5 cm. in length and 0.8 cm. in diameter. It connected the cephalic side of the bifurcation of the pulmonary artery with the concave side of the aorta just distal to the origin of the left subclavian artery (fig. 1,2). The dilated portion of the aortic arch was approximately twice the diameter of the ascending aorta. The aortic end of the ductus was found to be covered by a thin diaphragmatic membrane in which there were two openings, 1 to 2 mm. in their greatest diameter.

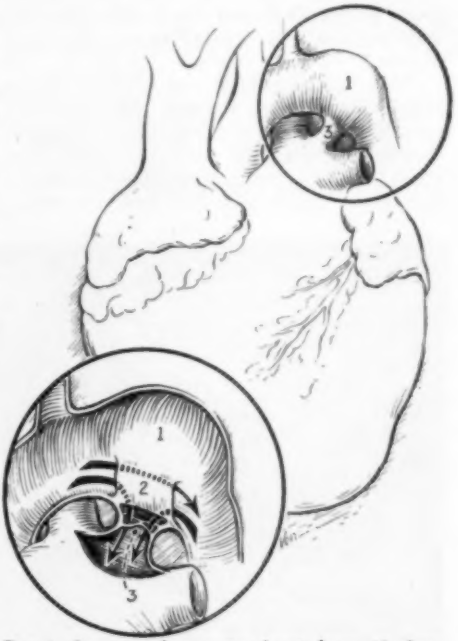

Fig. 1—Drawing of a canine heart (case 1) showing patent ductus arteriosus and aneurysmal dilatation of the aortic arch with an embryonic septal remnant; (1) dilation of the aorta, (2) intra-aortic septum, and (3) patent ductus arteriosus.

A second thin membranous septum, 1.5 cm. long by 1 cm. wide, was found attached to the concave side of the wall of the aorta (fig. 1). The distal end of this second mem-

From the Animal Hospital, University of Illinois, Chicago Professional Colleges, Chicago, and the Department of Surgery, College of Medicine, University of Illinois, Chicago.

brane formed a valvelike structure which has been reported to exist normally in the fetal and newborn dog and rabbit at this site.[6] However, the proximal end of the membrane formed one side of an opening between the aorta and ductus, with the wall of the aorta forming the other side of the opening.

Case 2—Patent Ductus Arteriosus with an Embryonic Septal Remnant.—During the course of a routine necropsy of a dog, a patent ductus arteriosus was discovered. The most interesting feature was a persistent septum attached to the concave side of the wall of the aorta with openings at both the proximal and distal ends (fig. 3). This septum was similar to the one seen in case 1.

Case 3—Tetralogy of Fallot.—This mixed breed dog, 3 years old, appeared normal. The chest was opened in preparation for experimental cardiac surgery. The heart was enlarged and spherical in shape. A systolic thrill was felt over the main trunk of the pulmonary artery and its branches, both of which were greatly dilated. This thrill disappeared upon obliteration of the orifice of the pulmonary valve by digital invagination of the wall of the dilated main trunk. Digital exploration of the pulmonic valve through the pulmonary artery revealed a strong systolic jet being ejected through the orifice of the pulmonary valve.

The mean pressures in the right ventricle and in the main pulmonary artery, taken through a 15-gauge needle attached to a mercury manometer, showed 46 mm. Hg in the right ventricle and 26 mm. Hg in the main pulmonary artery. After completion of the pressure measurement, the animal was euthanized.

A longitudinal incision into the pulmonary artery revealed a stenotic bicuspid valve with a slitlike orifice, 3 mm. in its largest dimension. The components of the dome-shaped valve were slightly thicker than normal leaflets. The dilatation of the

Fig. 2—The heart of a dog (case 2) showing a probe through the patent ductus arteriosus (1), demonstrating the intra-aortic septum (2), revealing free margins of the septum (3,4). A probe (5) is also passed through the aortic arch.

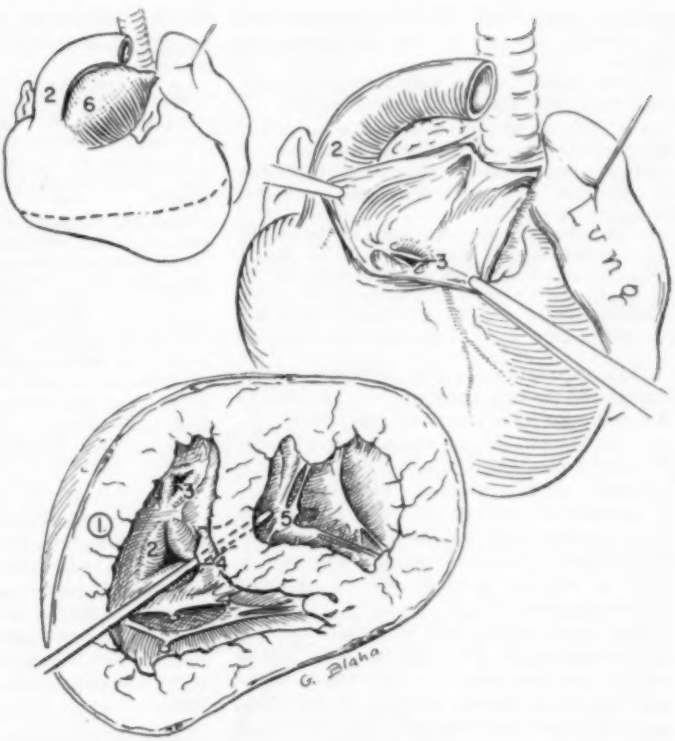

Fig. 3—A drawing of the heart of a dog (case 3) showing the tetralogy of Fallot; (1) hypertrophy of the right ventricle, (2) dextroposition of the aorta, (3) stenotic pulmonary valve, (4) interventricular defect, (5) medial leaf of the left A.V. valve in the left ventricle, and (6) poststenotic dilatation of the pulmonary artery.

main trunk of the pulmonary artery involved both main branches as well. The wall of the artery was thinner than usual.

Transection of the heart at the level half way between the atrioventricular groove and the apex disclosed the following additional abnormalities:

1) The wall of both ventricles and the inter-

Fig. 4—Photograph of a transverse section of a canine heart (case 3) revealing tetralogy of Fallot, showing hypertrophied wall of the right ventricle (R.V.), dextroposition of the aorta (A.O.), the pulmonary artery (P.A.O.), the tricuspid valve (T.V.), the left ventricle (L.V.), and a probe passing through the interventricular septal defect (I.V.S.D.) into the aorta.

ventricular septum was 1.5 cm. in thickness, and the papillary muscles in both ventricles were thickened.

2) The chamber of the right ventricle was approximately twice the size of that of the left.

3) A high dorsal interventricular septal defect, approximately 1.2 cm. in diameter, was found. It was funnel-shaped with its base opening to the left ventricle, and its apex located just below the aortic ring.

4) The aortic orifice was found to be in the right ventricle directly above the orifice of the interventricular septal defect and approximately 2.0 cm. caudal and 1.5 cm. dorsal to the orifice of the pulmonary artery (fig. 4). The aortic valve was tricuspid and the aortic orifice was normal in size.

This case presents the elements of tetralogy of Fallot, namely, (1) pulmonary stenosis, (2) interventricular septal defect, (3) dextroposition of aorta, and (4) hypertrophy of right ventricle. While the hypertrophy of the right ventricle can easily be explained as a result of the former three malformations, the huge dilatation of the main pulmonary artery was considered to be the product of the valvular pulmonary stenosis.

The blood in the left ventricle was entirely shunted to the right ventricle during systole. In the existence of such a shunt and mixing of blood, cyanosis is expected; however, none was noticed. The absence of noticeable cyanosis might be explained on the following hypothetical basis. The aortic orifice (fig. 4) is just above the right end of the interventricular septal defect. These two are connected by a deep groove which is not covered on the inferolateral aspect.

As the ventricles contract during systole, the gap between the aortic orifice and the right end of the interventricular septal defect will be shortened, decreasing the size of the defect and the amount of the shunt (fig. 4). This may also explain the absence of arterialization of the right ventricle.

DISCUSSION

The discovery of two congenital heart defects in 250 animals represents an incidence of slighty less than 1 per cent. Each lesion could have been diagnosed with simple auscultation and would then have offered an opportunity for study *in vivo* and surgical repair in either a surgical training or a surgical experimental program. The question is raised concerning the advisibility of routine physical examination of dogs used in an experimental laboratory.

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Effect of Maternal Anxiety on Offspring.—Since the injection of hormones such as cortisone, adrenalin, and ACTH into a pregnant female can have drastic effects on the fetus, strong emotion which would release such substances may have the same effect. That this could occur is indicated by tests on rats.—*Science (April 12, 1957): 698.*

Crying of the Fetus in the Uterus.—While crying does not necessarily indicate fetal suffering, the entrance of air into the uterus is an indispensable condition. When a woman was given an intra-amniotic injection of 700 cc. of oxygen, to visualize the placenta, the fetus started crying. It was audible 20 ft. away and was repeated at intervals for 50 minutes. It was reproduced on a phonograph record. The child was normal at birth 21 days later.—*J.Am.M.A. (Feb. 9, 1957): 499.*

An infant delivered by cesarotomy, 11.5 minutes after its mother's death, survived and progressed normally.—*J.Am.M.A. (March, 16, 1957): 938.*

The cornea of the eye can be preserved for two years at room temperature if first dehydrated in glycerine. This process does not cause cell damage as did the freezing process previously attempted. The demand for cornea transplants in man has exceeded the supply.—*Sci. News Letter (April 20, 1957): 249.*

Poisoning of Cattle with *Gutierrezia Microcephala*, a Perennial Broomweed

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LIVESTOCK owners have long suspected that the broomweeds are poisonous. Feeding tests[1,2] with two species of perennial broomweed failed to demonstrate toxic effects. Field observations suggested that, under certain conditions, the broomweeds were responsible for livestock losses. In 1933, feeding tests[3] with *Gutierrezia microcephelia* gave negative results. In 1934, the results were quite different: a 400-lb. steer died in three days after being fed 24 lb. of broomweed; a sheep died in five days after being fed 8 lb.; and a goat died in 14 days after being fed 11 lb.

Sporadic losses from abortion and premature calves in grazing cattle in the Trans-Pecos area of Texas reach alarming numbers in some herds in certain years. This condition has been a major problem in the southern parts of the High Plains area of Texas for approximately 50 years. It also occurs in other parts of Texas and New Mexico. The annual financial loss in Gaines County is estimated to be between $250,000 and $500,000. In affected herds, 10 to 60 per cent of the calves are premature or dead or weak at birth, weighing from 7½ to 50 lb. Some of the weak calves survive, if properly cared for, but most of them die before they are 48 hours old. Retained fetal membranes usually are associated with these abortions and occur in some cows that deliver apparently normal calves. Occasionally, a portion of the membrane will pass and the calf dies but is retained and must be forcibly removed. Deaths of cows are common, due to these events or to broomweed poisoning, or both. A survey of the grazing habits of such cows always shows them to be grazing broomweed.

In February, 1956, near Marathon, Texas, signs of broomweed poisoning occurred in 150 cows which were grazing broomweed in one pasture on a large range. Of the 150, 16 became critically ill and 4 died; 16 others showed some signs of intoxication. Although the illness occurred during a snowstorm, there had been warm weather previously and the leaves on the weed were growing rapidly. A few weeks later, a cow was observed with signs of broomweed poisoning on a ranch southeast of Marfa, where the cattle were grazing broomweed extensively. The rancher reported finding 6 small calves that were dead at birth or that had died shortly thereafter.

BOTANICAL DESCRIPTION

The species of broomweed incriminated, *Gutierrezia microcephala*, is a perennial herbaceous plant with a woody base and basal stems that become rootstocks when covered. The stems are corymbosely branched with few, if any, leaves on the lower parts (fig. 1, 2). The narrow, glabrous leaves are up to 45 mm. long and have a sticky exudate with a taste and smell similar to that of turpentine. The plant varies from almost prostrate under heavy grazing to erect and 3 ft. in height when growing under favorable conditions. Many small, yellow flowers are produced singly and in clusters up to three, with both disk and ray flowers brightly colored but fading with age. The involucre is about twice as long as it is wide and is top-shaped.

Gutierrezia microcephala occurs in central and western Texas; south into the states of Coahuila and Chihuahua, Mexico; west to New Mexico, Arizona, and Nevada; and north to Colorado, Utah, and Idaho.

POISONING EXPERIMENTS

Current studies were initiated in 1953 when field observations incriminated this plant as a possible cause of abortion.

Feeding Trials in 1953.—Plants, collected within a 20-mile radius of Marfa, were fed to 2 cows in April and to 5 from August through October (table 1). Plants were collected daily; the green portions were removed with pruning shears, ground

From Animal Disease Investigations, West Texas, Texas Agricultural Experiment Station, Marfa.

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The histological studies were made by Hilton Smith, D.V.M., Ph.D., head, Department of Veterinary Pathology, School of Veterinary Medicine, Texas A. & M. College, College Station.

Fig. 1—Gutierrezia microcephala (perennial broomweed).

in a hammer mill, and then mixed with ground alfalfa, molasses, and a vitamin A-fortified, 20 per cent protein grain meal. The cows fed in April consumed 2 lb. of broomweed per day; those fed later were started on 2 lb. but the broomweed was increased to 8 lb. daily within 18 days.

Feeding Trials in 1954.—In 1954, the feeding trials were conducted at Marfa in essentially the same manner as those in 1953. The weed was fed to 7 cows from March 1 until July 3. All had normal calves after being on this ration for 51 to 106 days and after consuming from 275 to 571 lb. of broomweed. Only 1 cow retained the fetal membrane (for 3 days). She had eaten 571 lb. of broomweed in 96 days. Four cows, fed as controls and receiving the same ration except for the broomweed, calved normally.

Feeding Trials in 1955.—Since losses occur almost every year in the Gaines County area, the feeding trials in 1955 (table 2) were conducted at Seminole, Texas. Observations of losses in that area implicated a number of plants. Those most consistently suspected were perennial broomweed and sand sage (*Artemisia filifolia*). These, both alone and in combination, and several other plants were fed.

In the area are two distinct soil types called "sandy land" and "hard land." The sandy soil is a "brownfield" fine sand 24 inches, or more, thick over a red sandy clay loam, which may be several feet thick; it is noncalcareous. The hard land is a "mansaker" fine sandy loam, a shallow soil with

Fig. 2—Typical area of Gutierrezia microcephala growing on loam soil.

TABLE 1—Broomweed Feeding Experiments, 1953

Cow No.	Plant fed (lb.)	Feeding period (days)	Results
4771	20	25	Weak, live calf, 35 lb.; retained fetal membrane.
4774	22	12	Dead calf, 40 lb.; dark urine; retained membrane.
4778	171	67	Calf (35 lb.) lived 30 hr.; retained membrane for 10 days.
4779	257	69	Calf (40 lb.) lived 48 hr.; retained membrane for 8 days.
4780	236	69	Live calf, 45 lb.; retained membrane for 9 days.
4782	159	29	Calved normally.
4783	107	24	Calved normally (60 lb.).

spots 10 inches, or less, to rock *caliche;* it is mildly calcareous and will be called "loam soil." Broomweed grows much more abundantly on the loam soil. During the first part of the feeding trials, the broomweed was gathered from the loam soil; but after observation showed that most of the losses occurred on sandy soil, broomweed grown on it was used.

Feeding trials were started in early January and continued through May. Weeds were collected as in 1953 and 1954 and mixed with ground hegari bundles and a vitamin A-fortified, 20 per cent protein meal. After finding that the blood serum levels of a number of cows that had delivered premature calves on the range were 15 to 43 μg. of vitamin A per 100 ml. of blood, the vitamin A meal was discontinued on February 20. Thereafter, the cows received only weeds, ground hegari bundles, a mineral salt, and water. The mineral salt contained dicalcium phosphate, defluorinated phosphate, cane molasses, iron oxide, manganese sulfate, vitamin D-activated plant sterol, and traces of cobalt sulfate, potassium iodide, and anise oil.

Feeding Trials in 1956.—Feeding trials were started, at Seminole, on January 26 and concluded May 7. Broomweed was collected from sandy soil and fed in the same way as in 1955. A total of 41 cows were fed broomwood at irregular intervals in an effort to learn at what stage of growth the plant is most toxic. Control cows in each group were fed the same ration, except for the broomweed. Some of the 14 control cows were fed sand sage.

Calves that weighed 50 lb. or more at birth and lived are considered normal in this report. However, some of these calves did not show normal strength and only 8 weighed as much as the smallest control. While normal Hereford calves in this area weigh 60 lb. or more (av. 70 lb.), only 4 cows fed broomweed from sandy soil delivered calves weighing as much as 60 lb.

Of 23 cows in a sandy soil broomweed-feeding experiment, 7 calved normally. After consuming 15 to 826 lb. of the weed in 5 to 95 days, their calves weighed 38 to 74 lb. Four cows which had normal calves (wt. 55 to 67 lb.) retained the fetal membranes; they had eaten up to 415 (av. 257)

TABLE 2—Feeding Experiments, 1955, with a Vitamin and Mineral Fortified Ration

Cow No.	Plant fed (lb.)		Feeding period (days)	Results
			BROOMWEED	
4935	917		99	Live calf, 62 lb.; retained fetal membrane for 8 days.
4940	696		80	Calf (40 lb.) lived 28 hr.; retained membrane for 10 days.
4950	1,097		117	Live calf, 41 lb.; retained membrane for 5 days.
4955	793		95	Live calf, 61 lb.; retained membrane for 5 days.
4965	54		21	Dead calf, 41 lb.; retained membrane for 3 days.
			SAND SAGE	
4933	440		62	Calved normally.
4951	1,257		117	Calved normally (60 lb.).
4952	1,453		124	Calved normally.
4964	331		65	Calved normally.
			BROOMWEED AND SAGE	
	Broomweed	Sage		
4937	181	239	38	Calved normally.
4945	689	665	112	Calf (37 lb.) lived 9 hr.; retained membrane for 10 days.
4947	303	387	87	Calf (31 lb.) lived 22 hr.; retained membrane for 5 days.
4949	193	255	40	Normal calf; retained membrane for 5 days.

Three cows, 1 fed cutleaf germander (Teucrium laciniatum), 1 fed scarlet gaura (Gaura coccinea), and 1 fed plains blackfoot (Melampodium leucanthemum), each calved normally.

Fig. 3—Cow showing mucopurulent nasal discharge and sloughing of the skin on the muzzle after being fed 10 per cent of broomweed per pound of body weight for eight days.

lb. of the weed in ten to 53 days. One of these cows died four days after calving. One cow, after eating 244 lb. of the weed in 38 days, died before calving (fetus wt. 50 lb.).

The remaining 11 cows gave birth to dead (8) or weak (3) calves and retained the fetal membranes. One of these cows died four days after aborting a 23-lb. calf. Of the 8 dead calves, 6 weighed 23 to 56 lb.—their dams had eaten 54 to 334 lb. of the weed in 11 to 57 days; while 2 weighed 7.5 lb.—their dams had eaten only 29 and 33 lb. of weed in seven and 13 days, respectively. The 3 calves which died, all in less than 48 hours, weighed 19 to 45 lb., and their dams had eaten 48 to 405 lb. of the weed in ten to 51 days.

Twelve other cows were fed sandy soil broomweed but did not calve during the course of the experiment.

Seven control cows fed from 62 to 755 lb. (av. 314) of sand sage in 18 to 83 days (av. 43) all had normal calves weighing from 55 to 80 lb. (av. 69) and all "cleaned" normally, except 2 that retained a small part of the fetal membranes for six and seven days, respectively. Seven other cows, used as controls, did not calve during the course of the experiment.

INFLUENCE OF SOIL TYPE ON TOXICITY

The unanimous opinion of ranchers in the Gaines County area that most of their abortions occur in cattle grazing on sandy land was confirmed. On Feb. 15, 1956, feeding trials were started to determine the difference in trouble caused by feeding broomweed growing on sandy soil and on loam soil. Six cows, five to seven months' pregnant, were fed weeds from each soil type. Those fed loam soil broomweed (227

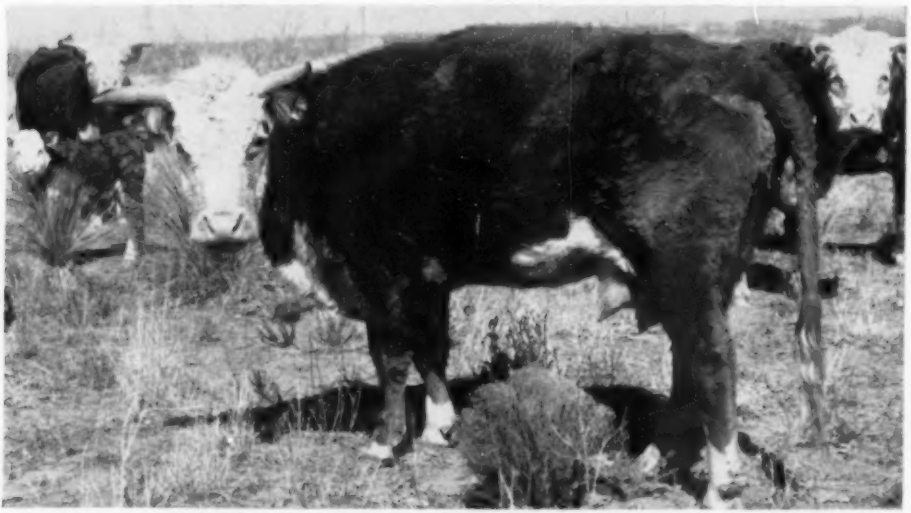

Fig. 4—Range cow showing typical signs of recent abortion and retained fetal membranes. Notice enlarged udder, also the broomweed plant in the foreground.

to 667, av. 558 lb.) over 74 to 82 days (av. 80) all had live calves weighing 41 to 66 lb. (av. 53), and only 1 retained the fetal membrane (3 days). Of the 6 fed sandy soil broomweed (236 to 541, av. 469 lb.) over 57 to 81 days (av. 66), 1 died before calving (41-lb. fetus), 1 had a live (47-lb.) calf but retained the fetal membrane, and 4 had dead calves (19 to 45 lb.) and retained the membranes.

ACUTE POISONING

In September, 1954, a 375-lb. heifer was forced fed broomweed (38 lb. in 14 days), collected in the vicinity of Marfa, and died on the nineteenth day. In 1956, a 730-lb. cow died 13 days after being forced fed with weeds (73 lb. in 8 days) collected in a pasture near Marathon, Texas, where cattle were affected. The leaves and 5 to 6 inches of the tops and branches were fed, excluding the woody and dry portions of the stems.

A number of cows became ill and lost weight prior to calving. Two of these cows died prior to calving. One, weighing 790 lb., died after being fed 527 lb. of broomweed in 81 days; the fetus weighed 41 lb. Another cow, weighing about 1,000 lb., died after eating 244 lb. in 38 days; the fetus weighed 50 lb.

Clinical Characteristics.—Acute poisoning by broomweed presents a different clinical picture from that usually seen in cows prior to abortion. Cows eating the more toxic broomweed develop periodic mucopurulent nasal discharge and the skin of the muzzle becomes crusted and sloughs (fig. 3). They also lose their appetite, lose weight, have a listless attitude, and a rough coat. Occasionally an animal will have dark brown or reddish urine. Animals urinate frequently and twist as if urination causes pain. Diarrhea, observed in early stages, later changes to constipation with large amounts of mucus in the foul-smelling feces. In addition, pregnant cows will have periodic vulvar swelling and, in most cases, an earlier-than-normal udder development. Hereford cows, which usually do not have large udders, often develop painful engorgement of the udder before aborting (fig. 4).

Gross Pathology.—Although there is no clinical evidence of icterus, the subcutaneous fat and connective tissue are light yellow to golden orange. The liver is soft and friable, the capsule peels off easily, and there are often pinpoint hemorrhages under the capsule and areas of apparent focal necrosis. The gallbladder shows inflammation of the mucous membrane which is stained yellow by the thick yellow bile. The kidneys and spleen are congested, and the pulp of the spleen is darker and softer than normal. There is a mild-to-severe gastroenteritis. The fetal membranes are usually edematous.

Histopathology.—The heart showed myocardial degeneration; the liver—congestion, fatty degeneration, and necrosis; the spleen—congestion with severe hemosiderosis and atrophy of the red pulp; and the kidneys—severe toxic nephritis with marked cloudy swelling, considerable cytoplasmic degeneration of the epithelium of proximal and distal convoluted and ascending tubules, with well-marked central displacement of their nuclei. There was congestion and edema of all layers of the uterus. The fetal membrane showed hydrops of the chorionic epithelium and a high percentage of binucleated and multinucleated syncytial cells.

DISCUSSION

The cows that aborted and retained the fetal membranes were tested and found negative for brucellosis and leptospirosis. Their blood serums were found to contain normal levels of vitamin A, and none had clinical signs of any other infectious disease.

Abortions and retained fetal membranes have been produced with as little as 20 lb. of broomweed and within seven days; at other times, it has taken as much as 1,097 lb. of broomweed and 117 days. Other cows have been fed 826 lb. in 95 days without producing any trouble. There is apparently a difference in susceptibility of individual cows. In spite of the fact that this species of broomweed grows more abundantly on loam soil, only sporadic losses are reported on this soil type. It has been observed that, where ranchers report losses regularly, such losses always occur on sandy soil.

During the feeding trials in 1956, many of the cows became critically ill and lost weight following calving. Two cows died on the fourth day following calving. This may have been due to retained fetal membranes and secondary infection or a combination of broomweed intoxication and infection.

Broomweed is apparently more toxic

when the plant is in a high rate of metabolism. Heaviest losses occur on ranches when the leaves are growing rapidly. Abortions were produced in experimental cows with smaller quantities of the plant at this stage of growth.

CONCLUSIONS

Broomweed, at some stages of growth, is toxic for cattle and will cause death, abortion, premature weak calves, and retained fetal membranes. The plant varies in toxicity at different periods and when growing on different soil types. Broomweed growing on sandy soil will cause abortion and retained fetal membranes more readily than that growing on loam soil.

SUMMARY

In four years, a total of 58 cows have been fed broomweed over varying periods of time in an effort to determine the abortifacient properties of this plant. The plant was ground, mixed with ground hegari or alfalfa, and fed free choice. Of the 58 cows, 29 (50%) aborted or delivered small weak calves, 35 had retained fetal membranes, and 5 cows died. Only 33 calves were raised.

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Rhabdomyosarcoma in the Heart of a Chicken

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Rhabdomyoma and rhabdomyosarcoma, the striated muscle tumors, are among the rare neoplasms of chickens.[1] In 1927, a tumor was described[2] that arose in the hip joint of a rooster, grew slowly, and proved transplantable. Rhabdomyomas of the pectoral muscle and submental region in chickens were discussed[3] and, in a survey of spontaneous neoplastic diseases in chickens, 2 cases which were diagnosed as rhabdomyoma were found.[4]

In man, tumors in the heart composed of rhabdomyoblasts are rare.[5] Whether these tumors should be referred to as true neoplasms has been questioned and the term "congenital nodular glycogenic tumor" was suggested to replace the term "congenital rhabdomyoma."[6] Benign striated muscle tumors or congenital glycogenic tumors have been observed in the heart of a guinea pig,[7] of swine,[8,9] and of a cow.[10] No reports have been found concerning the malignant form in the hearts of animals, including chickens.

This report concerns a striated muscle tumor that was observed at necropsy in the heart of a chicken and that has been diagnosed as a rhabdomyosarcoma.

HISTORY

A young capon which, 3 weeks previously, had received 15 mg. of stilbestrol for experimental purposes was presented for necropsy. It weighed 400 Gm. less than the average test birds at completion of the experiment.

GROSS PATHOLOGY

Necropsy revealed an emaciated capon but no other external anomalies. The heart, the only internal organ in which gross pathological changes were observed, was considerably enlarged and distended with marked dilatation of the right side. The walls of this side were thin and, when opened, an egg-shaped mass measuring 25 by 12 mm. was observed between the right atrium and the right ventricle on the wall of the interventricular septum and the septum atriorum. The mass greatly disturbed the morphological features of the atrioventricular opening and had obviously disrupted the normal valvular function. The valves themselves were thickened, and it was apparent that normal and complete closure of the valves had not occurred in the living specimen in the recent weeks before death.

The tumor had no pedicle but blended into the interventricular septum and septum atriorum. It was covered by endocardium, except for patchy areas of erosion, suggesting that initial growth had been either subendocardial or intramural. It was firm, somewhat darker than the heart muscle walls, and had several areas of necrosis

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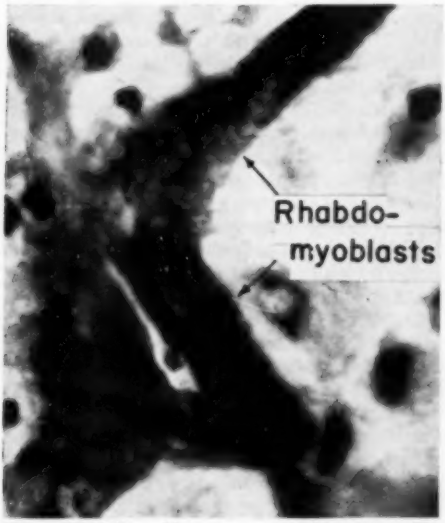

Fig. 1—Rhabdomyoblasts in an irregular pattern. The longitudinal striations are visible; cross striations are not apparent. H & E stain; x 800. Enlargement x 4.

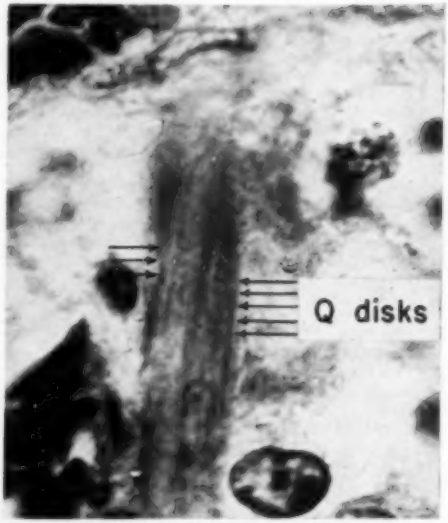

Fig. 2—Rhabdomyoblast with cross striations. Arrows point at Q disks. Gomori's one-step trichrome stain; x 800. Enlargement x 4.

on its surface. The cut surface revealed considerable ulceration principally at the periphery of the mass. The tumor offered little resistance to the knife and a distinct division between neoplasm and septums did not exist.

MICROSCOPIC EXAMINATION

Sections of the tumor stained with hematoxylin-eosin showed a gradual transition between normal cardiac fibers and tumor tissue. There were areas of inflammation with collections of polymorphonuclear neutrophils principally at the edge of the tumor away from the septums and, to a lesser extent, in the depths of the tumor and in the heart muscle in apposition to the tumor. There was some necrosis in both normal and tumor tissue. The neoplasm presented no definite morphological peculiarities, the cells being dispersed in an irregular sheet pattern. Gomori's one-step trichrome stain showed a few thin strands of collagen fibers that seemed to form the main supporting framework of the tumor. Blood vessels were numerous in the tumor tissue.

Vacuolation of the tumor cells, described in the literature as being characteristic of glycogenic tumors of the heart, was not noted in this tumor. The tumor cells were, for the most part, undifferentiated and those cells identifiable as rhabdomyoblasts were scattered irregularly throughout the tumor mass (fig. 1). The better differentiated rhabdomyoblasts were strap-shaped, acidophilic, but usually without cross striations. Cross striations were observed in a few isolated cells (fig. 2). The cells not recognizable as rhabdomyoblasts presented a great variety of sizes and shapes; double nuclei were numerous, whereas mitotic figures were rare. There were numerous cells ranging in size from 20 to 35 μ that showed variations in form from round-, to pear-shaped, to elongate-type cells. The elongate-type cells were the most acidophilic of this group and resembled rhabdomyoblasts more closely than did the paler round cells. This variation in form may have represented a transition from a poorly differentiated cell to a cell that bore some resemblance to the original embryonic myoblast. The tumor was invasive, for tumor cells were seen extending into and separating heart muscle fibers.

SUMMARY

A malignant tumor composed essentially of neoplastic striated muscle elements and growing from the interventricular septum and septum atriorum into the right atrium and ventricle of a young capon has been described.

The tumor was, for the greater part, poorly differentiated, although some rhabdomyoblasts with cross striations were observed. It was invading normal heart muscle at the point of its attachment to the septums.

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Neurofibromatosis or von Recklinghausen's Disease in a Dog

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Of the various types of tumor which may arise from the components of nerves, the majority are composed of the perineural fibrous tissue and nerve fibers. In some of these, the nerve fibers may predominate (neuroma); in others, the Schwann cells (schwannoma); in still others, the endoneural (endoneuroma) or perineural (perineuroma) structures predominate.[1] The following case represents one of the more generalized types called neurofibromatosis or von Recklinghausen's disease.

A spayed female Boxer, 2 years old, was presented for treatment on Oct. 15, 1956. Although she was emaciated, she had a good appetite, a normal temperature, and the feces showed no evidence of parasitism. A solid mass was palpated in the bladder region, the liver seemed enlarged and indurated, and fluoroscopy revealed a large growth in the thoracic cavity.

After euthanasia, a serous cyst, about 10 cm. in diameter, was found in the thoracic cavity. The liver also contained a large cyst which burst on removal of that organ. Another large mass, found near the bladder, contained a fibrinous, caseous material. The viscera and their contents were submitted to a pathology laboratory for examination.

Histologically, the tumor tissues proved to be neurofibromas, a condition sometimes called von Recklinghausen's disease in man. It is usually considered to be a disease of the peripheral nervous system. The central soft areas of necrosis were of a serous, degenerative type, probably because of the lack of blood supply to the center of each mass.

In von Recklinghausen's disease, the tumors are encapsulated, soft, fibrinous, sessile, or pedunculated. Minute nerve fibers are found entering and leaving the tumor. Occasionally, an elongated, tortuous proliferation of both the axis cylinder and sheath are found.

Neurofibromas consist of bundles of collagen fibers frequently showing myxomatous degeneration. Persons affected only occasionally report pain or paresthesia. The less common, larger neurofibromas, which are found in the peripheral nerves, may become painful and one or more of the growths may metastasize.[1]

This case is reported because it indicates how intriguing tumors can be. They should be submitted to a laboratory for diagnosis.

Dr. Antin is a small animal practitioner in New York City.

The author thanks Dr. Alexander Slawson and Dr. John M. King, Diagnostic Laboratory, New York State Veterinary College, for securing a diagnosis.

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Effect of Tarred Floors on Young Pigs.—Pigs fed pulverized floor slabs and cement containing coal tar developed signs and lesions similar to those in naturally poisoned animals. Those fed asphalt floor slabs and cement not containing tar remained healthy. Bitumen-containing, acid-resistant floor slabs are recommended for hog houses because of their insulating properties, provided they are free of tar.—*Vet. Bull. (March, 1957): 145.*

Antibiotic Therapy Can Increase Susceptibility to Salmonella Infection.—Mice treated with streptomycin the day prior to inoculation (both by stomach tube) were infected by a much smaller number of *Salmonella enteriditis* than is required to infect untreated mice. The antibiotic apparently affected the normal microorganisms which hinder the establishment of Salmonella.—*Science (April 19, 1957): 749.*

In Vitro and *In Vivo* Tests of Polymeric Piperazine-1-Carbodithioic Acid as an Equine Boticide and Anthelmintic

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THE TRIALS, using polymeric piperazine-1-carbodithioic acid (Safersan*) described here were initiated in an attempt to find a convenient, effective, and safe boticide.

This compound is a combination of carbon bisulfide and a piperazine base. In 125 mg. of polymeric piperazine-1-carbodithioic acid there is 150 mg. of piperazine (expressed as hexahydrate) and 58 mg. of carbon bisulfide. This compound is neutral in reaction and is stable except in suitable acid mediums when carbon bisulfide is evolved.[1]

The value of piperazine compounds as veterinary anthelmintics against a wide variety of worms is well established.[2,3]

Piperazine citrate, piperazine adipate, and piperazine phosphate were found to be equally effective anthelmintics if the dosages were adjusted to contain the equivalent of 200 mg. per kilogram of piperazine base (expressed as hexahydrate.) Also, *in vitro* polymeric piperazine-1-carbodithioic acid had boticidal properties but *in vivo* results were disappointing. In critical tests in horses,[3] piperazine adipate was 100 per cent effective against mature and immature *Ascaris equorum* and Trichonema spp., 50 to 60 per cent effective against *Strongylus vulgaris* and Triodontophorus spp., 80 per cent effective against *Oxyuris equi* females, but not effective against Gastrophilus spp., Habronema spp., or Anaplocephala spp.

Since the activity of polymeric piperazine-1-carbodithioic acid depends on its decomposition by acid[1] and since the pH of horses' stomachs varies from 1.13 to 6.8,[4] it was decided to test this drug at various hydrogen ion concentrations within this range to ascertain the degree of acidity required to provide for the decomposition and evolution of sufficient carbon bisulfide for boticidal purposes.

METHODS

Maintenance of Larvae.—In March, a horse's stomach was obtained 12 hours after death. Numerous third-stage larvae of *Gastrophilus intestinalis* were still attached. The cardiac area of the stomach, which contained the larvae, was removed, washed, and placed in digestive fluid (HCl 38% 10 ml., pepsin 6 Gm., 1,000 ml. distilled water). This solution was renewed each day and the specimen refrigerated at 40 F. until required. These larvae remained alive for over 21 days.

IN VITRO TESTS

Strips of stomach wall, with 6 bots attached, were suspended in beakers containing a 200-cc. suspension of polymeric piperazine-1-carbodithioic acid in modified digestive fluid at various concentrations and acidities. (The pH was altered by varying the proportion of hydrochloric acid). All experiments were carried out at 32 C. (table 1).

Death of the larvae was considered to have occurred when they became detached, ceased to move, and no heart beat was observed on microscopic examination following dissection.

During this experiment, the characteristic odor of carbon bisulfide was perceptible. To ascertain if carbon bisulfide gas or some soluble product of polymeric piperazine-1-carbodithioic acid was responsible for the boticidal action, a portion of stomach wall with 10 bots attached was suspended in a closed beaker above the surface of 200 ml. of a 1:200 suspension of polymeric piperazine-1-carbodithioic acid in modified digestive fluid at pH 1.3. The larvae became detached in less than ten minutes and were found to be dead. To ascertain if any of the soluble products of the compound had boticidal properties, the remaining liquid was then aerated until no odor of carbon bisulfide was perceptible and another portion of stomach wall with larvae was immersed therein. The larvae remained attached and active for over 18 hours.

Results.—The above findings indicated that, *in vitro*, efficient boticidal action of polymeric piperazine-1-carbodithioic could be obtained in a medium whose pH was 2.0 or less and where the concentration of the compound was at least 1:500 at pH 1.3 to

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*Boots Laboratories.

TABLE 1—In Vitro Tests with Gastrophilus Intestinalis in Various Concentrations of Piperazine-l-Carbodithioic Acid

pH	Concentration of drug and time required to kill larvae					
	1:100	1:200	1:300	1:500	1:1,000	1:2,000
1.3	...	3 min.	...	40 min.	...	...
1.8	...	10 min.	...	40 min.	10 hr.	*
2.0†	...	10 min.	30 min.	*	...	...
2.5	10 hr.	12 hr.	*	...	...	...

Larvae in all controls showed movement after 18 hours. *No effect movement of larvae seen after 18 hours. †At pH2.0 and concentration of 1:400, there was still movement of larvae after 18 hours.

pH 1.8 and 1:300 at pH 2.0. The results also indicated that the boticidal action was due to evolved carbon bisulfide. The inference was drawn that *in vivo* results would be disappointing unless measures were taken to procure a suitable acidity in the horse's stomach.

IN VIVO TESTS

Clinical trials with polymeric piperazine-1-carbodithioic acid were carried out on March 25, 1955, at Guelph, Ont. Three horses were used.

TABLE 2—Number of Bots Passed by Horses After Treatment with Polymeric Piperazine-l-Carbodithioic Acid

Horse (No.)	Dosage	G. intestinalis	G. nasalis
1	60 Gm.	106	45
2	60 Gm.	180	0
3	60 Gm.	62	18
4-12	None	0	0

Since bots are normally expelled in May and June, the feces from these horses were examined for several days before treatment and no bots were found. The feces of 8 control horses were also screened throughout the duration of the tests, and no bots were found. Therefore, it was considered that any expulsion of bots in experimental horses would be the result of dosing with polymeric piperazine-l-carbodithioic acid.

Palatability.—Polymeric piperazine-l-carbodithioic acid (60 Gm.) was mixed with bran and oats and given to the 3 horses, each weighing about 800 lb., which had fasted for 24 hours. However, because of unpalatability, none of the feed was eaten. Later, each of 5 other horses ingested 60 Gm. of the drug when the bran and oat mixture was covered with a thin layer of salt, and the horses' appetites were excited by rattling the feed pail.

Method of Dosing and Acidifying Horses' Stomachs.—After grain had been withheld from the 3 horses for 24 hours, during which time a small amount of hay was given, 60 Gm. of polymeric piperazine-l-carbodithioic acid was given by stomach tube, followed by about 200 ml. of water to remove any material remaining in the tube, and then by 300 ml. of 1:200 concentrated hydrochloric acid.

Results.—After dosing, 1 horse was slightly restless, but the other 2 showed no discomfort. Large numbers of strongyles were expelled between 24 and 72 hours after treatment. Bots did not pass until 48 hours after dosing but continued to be passed for five days (table 2). Movement was seen in only 3 of the bots passed. Fecal worm egg counts were made before and again five days after dosing (table 3).†

TABLE 3—Fecal Egg Counts Before and After Treatment with Polymeric Piperazine-l-Carbodithioic Acid

Horse (No.)	E.P.G. before treatment		E.P.G. after treatment	
	Ascaris	Strongyles	Ascaris	Strongyles
1	500	1,800	0	100
2	100	750	0	100
3	0	200	0	0

CRITICAL TEST

Ten days after dosing, 1 horse was killed and examined; all organs appeared normal.

Though 106 larvae of *Gastrophilus intestinalis* had been passed, 52 were still attached to the stomach wall (table 4). At the pylorus, there were 3 living larvae of *Gastrophilus nasalis*, and an extensive thickening was noticeable showing ulcers where other larvae had been attached.

The colon and cecum showed numerous bite marks but no small strongyles were found attached to the mucosa, although a few were found mixed with the feces.

The entire bowel contents were mixed and measured and samples examined for worms by dilution technique (table 4).

DISCUSSION

In vitro results, using polymeric piperazine-l-carbodithioic acid, indicate very lit-

†Information from Boots' Laboratories, received after submission of this paper, indicates that for complete dissociation of carbon bisulfide in vivo a greater quantity of HCl than that used in this experiment may be required.

TABLE 4—Result of Critical Test (Horse 1)

Nematodes	G. intestinalis	G. nasalis	Ascarids	Large strongyles	Small strongyles	Female O. equi	S. vulgaris
Expelled	106	45	..	0	18,400	28	25
Found at P.M.	52	3	0	36	700	0	20
Effective (%)	67.0	93.6	..	0	96.3	100	55

This test was performed ten days after giving this 800-lb. horse 60 Gm. of polymeric piperazine-1-carbodithioic acid followed by 300 ml. 1:200 HCl.

tle difference in boticidal effects at concentrations of 1:200 and 1:500 at pH 1.3 and 1.8.

At pH 2.0, while boticidal action was efficient at a concentration of 1:200, no effect was apparent after 18 hours immersion at concentration of 1:400.

At pH 2.5 and concentration of 1:200, death of the larvae did not occur until 12 hours after immersion and, at the same pH and concentration of 1:300, no effect was apparent after 18 hours.

This would suggest that in order to obtain the full advantage of rapid evolution of the carbon disulfide contained in polymeric piperazine-l-carbodithioic acid, a medium of minimum acidity of pH 2.0 is required.

In testing the *in vitro* effects of this compound against larvae of *G. intestinalis*, it had been found[3] that 1.5 Gm. polymeric piperazine-l-carbodithioic acid in 400 ml. of 0.2 per cent of HCl caused detachment of larvae from the mucosa in ten minutes. After three hours immersion, the fluid was removed and normal saline added. No reattachment occurred overnight. However, on dosing horses with polymeric piperazine-l-carbodithioic acid in November and December, no bots were expelled. This led to the conclusion, "There is little or no action against *G. intestinalis* in the stomach; either the compound is not completely dissociated or the liberated carbon disulfide does not reach the bot larvae. The horse's stomach has a rapid emptying time and this may well be related to the poor results obtained against bot larvae."[3]

The authors' findings are not at variance with those above. However, in view of the *in vitro* experiments, it is to be expected that sufficient liberation of carbon bisulfide would not occur unless the stomach pH of the experimental horse was 2.0 or less, (it varies from pH 1.13 to 6.8). Furthermore, those tests were carried out in November and December, whereas the present work was done in March.

Since the dose used was 60 Gm., the effects, if complete dissociation had occurred, should have been equal to that of 72 Gm. piperazine base (expressed as hexahydrate) and 27.84 Gm. of carbon disulfide.

These tests were carried out (March) when the Gastrophilus larvae were fully grown, although not yet ready to pass normally from the stomach. The dose of 300 cc. of 1:200 HCl was chosen arbitrarily.

The failure to find *A. equorum* in the feces was disappointing. The fecal egg counts recorded, however, indicate 100 per cent efficiency against *A. equorum*.

Should further tests confirm the usefulness of this drug in conjunction with acidulation, the main advantage derived would be in having a combined effective anthelmintic and boticide free from the hazards of administration which occur with carbon bisulfide liquid or capsules and with all the anthelmintic advantages of piperazine salts.

SUMMARY AND CONCLUSION

In vitro trials with polymeric piperazine-l-carbodithioic acid at various concentrations in mediums of different hydrogen ion concentrations, indicated that it was effective only at a minimum acidity of pH 2.0.

It was also determined that the boticidal action was due entirely to evolved carbon bisulfide.

In a limited clinical trial, in March, using polymeric piperazine-l-carbodithioic acid followed by hydrochloric acid, boticidal and anthelmintic effects were demonstrated.

Results of critical test on 1 horse indicated the following percentage of removal efficiency: *Gastrophilus intestinalis*, 67.0; *Gastrophilus nasalis*, 93.6; large strongyles, 0; small strongyles, 96.3; *Oxyuris equi* females, 100; and *Strongylus vulgaris*, 55.

Results by fecal egg counts indicated 100 per cent efficiency against *Ascaris equorum*.

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Piperazine Derivatives Have Tranquilizing Effects.—Research with laboratory animals indicates that piperazine derivatives affect the chemistry of the nervous system. They have a powerful blocking effect against drugs such as ephedrine and amphetamine but a relatively small effect on drugs such as adrenaline. In man, they produce drowsiness and, after two or three weeks of oral administration, they produce dreaming which is so frightening that the patient fears sleep. Mental confusion and lethargy often occur.—*Science (April 19, 1957): 721.*

Congenital Porphyria (Pink Tooth) in a Heifer

This rare metabolic disease of animals and man is due to a high level of porphyrins in the blood, with depositions showing in the teeth and bones and sometimes in the lymph nodes, liver, lungs, and other organs. There also may be skin lesions of photosensitivity as well as retarded growth and anemia. It has been reported in 13 cattle sired by 1 bull in South Africa; in pigs in New Zealand; in pigs and cattle in Denmark; and in cattle in the United States. It is most commonly recognized in slaughtered animals.

The case described occurred in a light roan Shorthorn heifer, 30 months old, which showed dry, yellowish gray, scablike areas along the back; striking, brownish pink teeth, mainly the dentine; anemic mucous membranes; and reddish brown urine. She had been raised indoors because in the springs of both 1955 and 1956, when turned on grass, she developed lesions of photosensitization along her back. She was stunted, and no permanent incisors had erupted. When slaughtered, all her bones were brown. A bright red fluorescence was seen when the teeth and bones were examined in ultraviolet light. No other abnormalities were found in the herd except that the dam had to be kept out of the sunlight since she became "sweaty along the back in hot weather."

Congenital porphyria is differentiated from acute idiopathic porphyria, a dominant inherited condition in man in which photosensitivity and pigmentation of the bone is absent; and from symptomatic porphyria which occurs in poisoning on lead, barbiturates, etc., but in which heredity, photosensitivity, and pigmentation are not factors.—*D. B. Ross in Vet. Rec. (March 16, 1957): 345.*

Resistance of Sheep Nematodes to Phenothiazine

The response of two strains of *Haemonchus contortus* to the action of small daily doses of phenothiazine presented striking differences that agreed closely with previous findings by the authors. Three experiments were completed, utilizing a total of 14 helminth-free lambs and daily dose levels ranging from 0.1 to 2.0 Gm. of the drug.

Strain B from a flock long treated because of a worm-control problem, not only had a threshold four to eight times higher than the control strain A in the present studies, but also appeared to have a higher phenothiazine threshold than strains of *H. contortus* studied by other investigators. Coupling the laboratory investigations with the flock history of strain B indicates that the prolonged and intensive use of phenothiazine has resulted in the selection or development of this strain of *H. contortus*, possessing a significant level of resistance to phenothiazine.—[*J. H. Drudge, S. E. Leland, Jr., and Z. N. Wyant: Strain Variation in the Response of Sheep Nematodes to Action of Phenothiazine. II. Studies on Pure Infections of Haemonchus Contortus. Am. J. Vet. Res., 18, (April, 1957): 317-325.*]

Treatment for Nasal Grubs in Sheep.—Because of the shielding action of mucus and exudates, the treatment for ovine nasal grubs has been ineffective. A formula containing 4 mg. of lindane in a combination of solvents and emulsificants was effective in doses of 4 cc. injected into each nostril. Of 2,865 affected sheep, most recovered in nine to 14 days and the balance (6%) recovered after a second treatment.—*DuToit and Fiedler in Onderstepoort J. Vet. Res., 27, (1956): 67-75.*

African Swine Fever in Wart Hogs (*Phacochoerus Aethiopicus*)

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IN HIS ORIGINAL publication on African swine fever (ASF), Montgomery[1] reported the presence of wild pigs, chiefly wart hogs, in the vicinity of every farm on which ASF infection had been diagnosed. Subsequently, other workers[2-4] succeeded in recovering the virus from apparently healthy wart hogs killed in areas where ASF had occurred in domestic swine. Although it is established that wart hogs can act as inapparent carriers of ASF virus, little is known concerning the mechanism of transmission from wart hog to wart hog or from wart hog to domestic swine.

LITERATURE CITED

The theory was advanced[2] that in nature ASF infection is maintained by the carnivorous habits of wild swine rather than through the agencies of contact exposure. It was also suggested[3] that it is transmitted in nature by means of an intermediate host, probably a winged insect.

A wart hog was infected by inoculation with ASF virus, but a second wart hog did not become infected when placed in contact with the inoculated wart hog and with affected domestic swine.[3] African swine fever viremia was demonstrated in an inoculated wart hog on postinoculation (PI) days 6, 9, 13, and 17 but not on PI day 21. Domestic pigs placed in contact with 2 known ASF-infected wart hogs did not contract the infection. Later, 2 wart hogs, infected by inoculation, failed to transmit the disease to domestic pigs by contact exposure.[5] It was concluded[5,6] that the serum of wart hogs possessed no protective properties.

During the 1954 outbreak in Kenya,[4] a wart hog sow and her pig were killed on the infected ranch. African swine fever virus was isolated from the sow but not from her pig. This finding could be considered as further evidence that ASF does not spread readily by contact among wart hogs.

However, the virus is maintained in a wart hog population for years by some means. The following experiment was designed to learn more about ASF in wart hogs and to attempt various transmission trials by contact exposures.

EXPERIMENTAL PROCEDURES

Three young wart hogs, 1 male and 2 females from the same litter (fig. 1, 2), were obtained in December, 1954. They were estimated to be between 4 and 8 months old and weighed between 30 and 40 lb. The proposed plan was to infect wart hog 1 by placing it in contact with ASF-infected domestic pigs, then to infect wart hogs 2 and 3 by placing them in contact with wart hog 1, and to complete the cycle by infecting domestic pigs by contact exposure with wart hogs 2 and 3. While the experiment did not go exactly according to plan, some additional information was obtained.

Blood specimens for viremia tests were obtained from the anterior vena cava of the wart hogs. Susceptible domestic pigs were then subinoculated with 5 ml. of this fresh wart hog blood to which 0.1 ml. of a solution containing 10 mg. of the sodium salt of heparin per milliliter was added. The test was considered positive if the subinoculated pig sickened and died of ASF; negative if the subinoculated pig remained normal and was later susceptible when challenged with ASF virus.

Before starting the transmission trials, a viremia test was made on the 3 wart hogs which were found to be negative.

Part 1.—Wart hog 1 was exposed by contact with 3 domestic pigs, which sickened and died of ASF between Feb. 23 and March 8, 1955. On March 7, wart hog 1 was positive to a viremia test. (While wart

Fig. 1—A male wart hog.

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Fig. 2—The wart hogs used in this experiment.

hog 1 was with the 3 infected pigs, the same thermometer was used on all 4 animals. Therefore, it could have been infected through the rectal mucosa.) Sixteen days later, wart hog 1 was again positive to a viremia test. Thus, the first step in the proposed plan was at least partially successful; wart hog 1 became infected without parenteral inoculation. However, it was negative for viremia on April 20.

Part 2.—On March 23, 1955, when wart hog 1 was known to have ASF viremia, it was placed in contact with wart hogs 2 and 3 and remained with them until July 8. Wart hogs 2 and 3 were negative when tested for ASF viremia on April 6 and 20. Two susceptible domestic pigs, which were placed in contact with wart hogs 2 and 3 on July 13, remained normal over an observation period of 22 days, and both were susceptible when inoculated with the virus at the end of this period. There was no indication that wart hogs 2 and 3 became infected through contact exposure to wart hog 1.

Part 3.—Since the second step in the proposed plan was unsuccessful, the 3 wart hogs were re-exposed by contact with 2 domestic pigs which sickened and died of ASF between Aug. 8 and 21, 1955. On August 25, 2 susceptible domestic pigs were placed in contact with the 3 wart hogs. No reaction occurred. On September 20, 2 more susceptible domestic pigs, No. 2237 and 2238, were placed in the same stall; 21 days later, pig 2237 sickened and it died of ASF five days later. The other 3 in-contact domestic pigs became ill and died between October 18 and 27, presumably from contact exposure to pig 2237. On November 10, the 3 wart hogs were negative for viremia. On November 28, 5 more susceptible domestic swine were placed in contact with the 3 wart hogs; no reaction occurred in 92 days.

Part 4.—On Feb. 28, 1956, the 5 domestic swine were isolated from the 3 wart hogs and each of the latter was given 4 ml. of virulent ASF virus blood, their first exposure by inoculation. On March 5, 4 domestic pigs were again placed in contact with the 3 wart hogs. They remained normal for 49 days and were susceptible when then challenged with ASF virus.

On April 23, 1956, the 3 wart hogs were killed and tissues, including blood, lymph nodes, salivary glands, kidneys, spleen, lung, brain, and bone marrow, were collected at necropsy. A 20 per cent suspension in 0.85 per cent NaCl was made of mixed tissues from each wart hog. Three susceptible domestic pigs were inoculated with 5 ml. of the suspension. The 2 pigs inoculated with tissues from wart hogs 1 and 3 remained normal and were susceptible when challenged later with ASF virus. The pig inoculated with tissues from wart hog 2 sickened and died, showing typical ASF lesions at necropsy. Thus, wart hog 2 was still carrying the virus in at least one tissue 54 days after it was exposed by inoculation, yet it did not transmit it to 4 domestic swine which were in the same stall for 49 of the 54 days.

Fig. 3—Wart hogs cohabitating with domestic swine.

TABLE 1—Protection Test with Wart Hog Serums

Test pig No.	Serum	ASF virus	First PI day of illness	PI day of death
2297	45 ml. from wart hog 1	100 $l.d._{50}$	6	13
2298	50 ml. from wart hog 2	100 $l.d._{50}$	16	20
2299	80 ml. from wart hog 3	100 $l.d._{50}$	Not sick	24 (Accidental)
2300	None (virus control)	100 $l.d._{50}$	7	16

PI=postinoculation.

Serum-Protection Tests.—Serum was removed from the blood collected at necropsy from the 3 wart hogs and was inactivated by heating at 56 C. for 30 minutes. Domestic pigs weighing 40 lb. each were inoculated simultaneously with wart hog serum and 1 ml. of a 10^{-4} dilution of the Kenya Hinde strain[4] of ASF virus. The serum was injected into the axillary spaces, and the virus (approx. 100 $l.d._{50}$) was injected behind one ear. It appeared (table 1) that serum from wart hog 2 delayed the illness and that serum from wart hog 3 prevented a reaction to the simultaneous virus challenge.

Test pig 2299 (table 1) died accidentally while being bled on PI day 24. This pig showed no signs of ASF other than transient febrile reactions on PI days 9 and 12 and no lesions were present at necropsy. Serum taken from pig 2299 on PI day 24 was used in a serum-protection test (table 2) after having been inactivated (30 min. at 56 C.).

Even though pig 2299 appeared to be protected by serum from wart hog 3, its own serum gave no protection against either simultaneous or contact exposure; its viremia test on PI day 24 was negative.

DISCUSSION

The host-parasite relationship between the wart hog and ASF virus is ideally suited for the survival of both. An equilibrium is apparently reached, which permits survival of the parasite virus without apparent harm to the host wart hog. The presence of ASF virus in Africa would probably have gone unnoticed had it not been for the introduction of an unnatural host, the domestic pig, into the environment. When the virus was transmitted from its natural host, the wart hog, to an introduced host, the domestic pig, it caused a contagious, peracute, and highly fatal infection.

Many questions remain to be answered concerning the epizootiology of African swine fever. Under experimental conditions the virus does not appear to spread readily by contact from wart hog to wart hog or from wart hog to domestic pigs. The contact infection to a domestic pig on Oct. 11, 1955, is the first recorded. Attempts to repeat this method of infection failed. When this successful contact exposure occurred, there were other ASF experiments in progress in the same isolation unit; therefore, accidental exposure to pig 2237 can not be entirely ruled out. However, during two years of work on ASF, there have been no other cases of cross-infection from one stall unit to another.

The short periods of viremia in wart hogs previously reported,[1,5] and in wart hog 1 reported here, do not seem consistent with the persistence of the disease for years in the wart hog population. Therefore, it was of considerable interest to recover the virus from wart hog 2 when it was killed 54 days after it was last exposed. Obviously, a negative viremia in wart hogs does not necessarily indicate the absence of infection since the virus might reside in some focal area without being present in the blood.

The suggestion[5] that a winged insect

TABLE 2—Protection Test with Serum from Domestic Pig 2299

Test Pig No.	Serum	ASF virus	First PI day of illness	PI day of death
2312	80 ml.	1,000 $l.d._{50}$	4	14
2313	80 ml.	Contact	14	20
2315	None (Virus control)	1,000 $l.d._{50}$	4	11
2316	None	None (Contact control)	8	17

PI=postinoculation.

might act as an intermediate host of ASF virus can not be excluded but events in recent years in Kenya would seem to minimize this possibility. Steps taken to reduce the opportunities for contact between wild and domestic swine appear to have been instrumental in reducing the incidence of the virus. From 1913 to 1944, before these isolation measures were implemented, 60 outbreaks of ASF were recorded; since 1944, only four have been reported.

At no time did the 3 wart hogs used in these experiments show ill effects from exposure to the virus. Their normal temperatures ranged from 97.6 F. to 103.4 F. with no increase following exposure. The wart hogs lived contentedly with domestic pigs after a few days of adjustment (fig. 3), during which they made a determined attempt to destroy their domesticated cousins. The caretaker had no trouble in feeding the wart hogs or in taking their rectal temperatures, once they were accustomed to him, but they never submitted to restraint for bleeding without a fight. Therefore, viremia tests were not made as often as might have been desirable. Once restrained on the bleeding trough, blood specimens were taken from the anterior vena cava without difficulty.

The apparent protection of domestic pig 2299 with serum from wart hog 3 was inconclusive. The serum may have been instrumental in giving protection or the pig may have had some innate resistance which permitted its survival to the relatively light virus exposure. The accidental death of pig 2299 was unfortunate because it prevented a subsequent challenge to determine whether or not this pig was immune to ASF. Since serum from pig 2299 failed to protect pigs against either an ASF simultaneous virus challenge or contact exposure and since this pig showed no viremia on the day of death, it likely would have sickened after a more concentrated, subsequent virus challenge.

SUMMARY

African swine fever (ASF) was not readily transmitted to domestic swine by experimental contact with known-infected wart hogs. However, one apparently successful transmission by this method occurred. African swine fever virus was recovered from a wart hog 54 days after it was exposed by inoculation. Previously, 17 days was the longest period confirmed for a wart hog to act as a carrier of ASF virus. Protection tests with wart hog serums in domestic pigs were inconclusive.

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Pleuropulmonary Tularemia.—Of 28 patients with tularemia, at a Maryland hospital in 8 years, pleuropulmonary involvement was demonstrated radiographically in 14. Of the 14, seven had the typhoidal form, five the ulceroglandular form, and two primary infections of the lung. In 9, the involvement of the pleura varied from a slight thickening of an interlobar septum to extensive diffusion. All responded to antibiotic therapy.—*J.Am.M.A. (April 6, 1957): 1304.*

Cats and Clostridium Botulinum Toxin.—It is suggested that the cat may be resistant to botulism but is a carrier of the organism and that the toxin is elaborated in the carcass after death.—*Ann. Inst. Pasteur, 89, (1955): 354-357; also in Vet. Bull., 26, (1956): 1895.*

[This could explain reports of botulism in animals fed forage which had been in contact with decomposed small animal carcasses.]

Infectious canine hepatitis virus has been successfully grown in tissue cultures of dog kidney epithelial cells. Virulence for the natural host was reduced by continued passage.—*Nature (Feb. 23, 1957): 420.*

Correction—Chlortetracycline and Nursing Lambs

In the article "Effect of Constant Intake of Chlortetracycline on Pneumonia, Enterotoxemia, and Production in Nursing Lambs" (April 15, 1957, JOURNAL, p. 351), the tenth line under Results and Discussion should read: *mg. per 1.0 lb. of body weight sought.*

Clinical Signs of Blastomycosis in Dogs

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GILCHRIST[5] was the first to report the occurrence of American-type blastomycosis in man in 1894. Therefore, the disease has often been referred to as Gilchrist's disease. In 1896, the causative organism was isolated from a second reported case and named *"Blastomyces dermatitidis."*[4] The microorganism was found to be unicellular, budding, and yeastlike in the tissues of man and other animals but often in mycelial form in culture.

The disease in man is either cutaneous or systemic.[4] Cutaneous blastomycosis consists of primary infections of the skin, although cutaneous and subcutaneous lesions may occur as a result of a hematogenous spread of the *B. dermatitidis* organism from a primary pulmonary lesion. Systemic blastomycosis usually occurs as a result of the entry of the organism by way of the respiratory tract. Secondary skin lesions, bone infection, and involvement of other organs and tissues of the body occur through the dissemination of the *B. dermatitidis* organism from the primary pulmonary lesions.

The present report is concerned with blastomycosis in animals, and the canine species in particular. Earlier reports[6,7] have shown that with the aid of more accurate diagnostic procedures and an increased interest in the disease itself, blastomycosis has been found to be much more prevalent in dogs in central Kentucky than had previously been realized. Infection in man has also been found to be more prevalent in this particular geographic region than elsewhere. However, this does not infer that one follows the other or that there is a definite association between dogs and man regarding the source or frequency of the disease.

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The authors express their sincere appreciation to Dr. J. T. McClellan, M.D., and Dr. A. Balows, Ph.D., pathology department and laboratory of the St. Joseph's Hospital and Lexington Clinic, Lexington Ky., for their cooperation, assistance, and advice regarding the pathological diagnosis of the disease from tissue specimens and exudates, also for the cultivation of the etiological agent from exudates and biopsy material. The authors also thank Dr. Libero Ajello, Ph.D., and his staff at the Mycology Unit, U. S. Public Health Service, Chamblee, Ga., for cooperating in the confirmation of the diagnosis by serological examination of suspected cases.

The following observations were made on 55 dogs which were found to be infected with the *B. dermatitidis* organism. The tentative diagnosis of blastomycosis was made either from the clinical manifestations of the skin lesions, or from the respiratory changes in the cases of systemic blastomycosis. The diagnosis was then confirmed by one or more of the following methods: (1) serum complement-fixation; (2) culture of the organism, *B. dermatitidis*, from necropsy or biopsy tissue specimens; and (3) identification of the fungus by microscopic examination of tissue or a characteristic exudate derived from a typical lesion.

There appeared to be considerable individual variation in the signs of the disease in animals but a large majority of the cases recognized were classified as follows: (1) chronic respiratory type; (2) skeletal type; and (3) acute generalized type.

Chronic Respiratory or Pulmonary Blastomycosis.—Of the 55 animals, 43 were affected with the chronic respiratory type. The first signs were usually an acute pneumonia accompanied by a temperature of 103 to 105 F. At this stage, clinical differentiation from typical pneumonia was difficult, if not impossible. The fever and accompanying signs persisted for approximately six days, followed by slight improvement. At this stage, the animals were often returned to their owners and not seen again for four to five weeks. However, many were readmitted for further examination and treatment chiefly because of an increased, persistent coughing.

In some animals, the first sign was a slight cough which increased in severity, causing owners to seek the help and advice of a veterinarian. The temperatures continued to be normal in these cases.

In the more or less acute forms of the disease described above, all of the animals eventually developed a long, dry, whistling cough with a definite stenotic bronchial sound at a point above the heart. The course of the disease in these patients was variable. For example, 1 dog was severely affected from October, 1950, to May, 1952,

but when examined in October, 1952, and June, 1954, it appeared to be normal.

Two other animals with blastomycosis, confirmed by complement-fixation, began coughing only with the onset of inclement weather. Other dogs were euthanized after several weeks' observation to terminate their increased suffering. In one of the latter, a fibrous mass involving the bronchial lymph nodes was so large that it not only interfered with respiration but also had displaced the esophagus against the thoracic wall, resulting in restriction of the passage of food.

For public health purposes, the authors have encouraged euthanasia of the affected animals. The owners frequently request this on their own initiative when given the facts. Therefore, the natural termination of these cases is not determined

Chest radiographs have been found helpful in the diagnosis of this disease. They usually show dense masses dorsal to the heart, indicating involvement of the bronchial lymph nodes. However, the diagnosis should be confirmed serologically. For example, 2 animals with blastomycosis-like lesions on radiographs were found on necropsy to have a thickening of the tracheal and bronchial tree due to an infection with *Filaroides osleri*.

Skeletal Blastomycosis.—Blastomycosis of the long bones, in 4 dogs, had been tentatively diagnosed as traumatic injuries and were treated as such. Each started as a small, sensitive, puffy swelling which eventually became extremely painful, even when palpated lightly.

These lesions were located as follows: (1) distal one third of the radius; (2) distal one third of the tibia; (3) between and involving both third and fourth metatarsals, distally; and (4) in the proximal portion of a radius and ulna.

Radiographs revealed areas of localized osseous rarefaction. Blastomyces were found on microscopic examination of all biopsy specimens. All 4 cases terminated in generalized blastomycosis within 13 to 70 days.

Generalized Blastomycosis.—This form of the disease often followed a lowered body resistance due to parasitism, parturition, lactation, exposure, and bacterial infections. Eight of the 55 affected dogs had generalized or systemic blastomycosis. Initially, the diagnosis was frequently confused with distemper. Some owners were critical of distemper vaccination results until blastomycosis was demonstrated at necropsy.

This form of the disease started with a temperature rise to 103 to 105 F., a purulent nasal discharge, cough, accelerated respiration, and a poor appetite. There was no response to antibiotic or other therapy.

The course of the disease varied from five to 30 days, depending upon the animals' general condition. Among the terminal signs observed were: (1) a glassy stare and miotic pupils; (2) severely congested, scleral, palpebral conjuntivae; (3) sanguinous nasal discharge; and (4) pronounced pneumonic signs. Two of these cases had multiple subcutaneous abscesses over the entire body (diam. 1 to 3 cm.). *Blastomyces dermatitidis* was demonstrated in the exudate from these lesions. In all cases, stupor and convulsions preceded de by a few hours.

SUMMARY

Four years of clinical experience with blastomycosis, in a small animal practice, indicates that the disease can be classified as acute and generalized, chronic respiratory, or skeletal in type. Clinical diagnosis may be based on characteristic respiratory changes, the appearance of typical skin lesions, and the demonstration, by radiographs, of abnormally dense masses dorsal to the heart. The diagnosis should always be confirmed serologically and by demonstrating the etiological agent in cultures from the exudates and lesions.

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What Is Your Diagnosis?

Because of the interest in veterinary radiology, a case history and radiographs depicting a diagnostic problem are usually published in each issue.

Make your diagnosis from the picture below—then turn the page ▶

Figure 1

History.—A male, German Short-Haired Pointer, 9 years old, experienced periodic attacks of diarrhea for three months but ate well and appeared normal. The attacks became more frequent and the straining increased at each bowel movement. Fecal examinations revealed no parasite ova. The stools were soft, not over 1½ cm. in diameter, and fresh blood and mucus frequently were seen on the surface.

Figure 2

The dog objected to rectal digital palpation, the area of the prostate being most sensitive. The bowel felt normal as did the prostate (3 cm. diam.) which was palpated on the brim of the pelvis. A proctoscope passed freely to a depth of 17 cm., where it met resistance. After an enema was expelled, the rectum was filled with barium solution and lateral radiographs were taken.

(Diagnosis and findings are reported on next page)

Here Is the Diagnosis

(Continued from preceding page)

Diagnosis.—A long, irregular filling defect of the posterior portion of the descending colon.

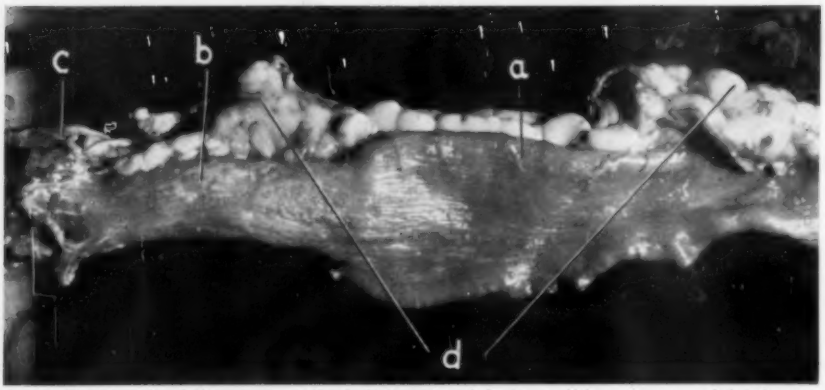

Fig. 3—Photograph of the serous surface of the colon (a), tumor (b), and rectum (c); also two involved lymph nodes (d).

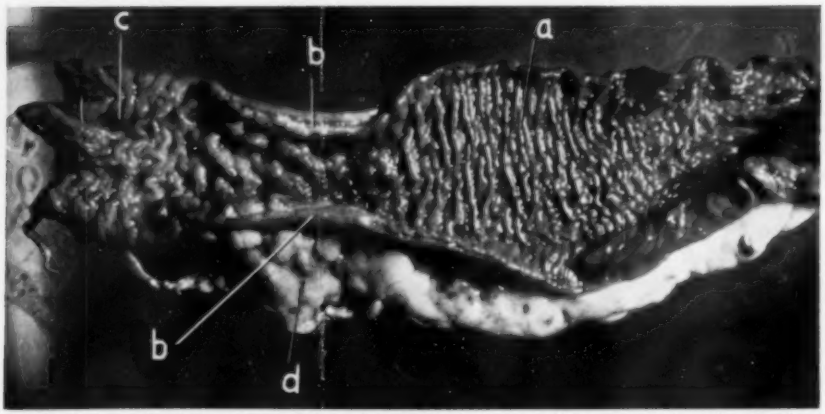

Fig. 4—Photograph of the mucous surface of the colon (a), tumor (b), rectum (c), and an involved lymph node (d).

Comment.—The scalloping seen on the barium film, in addition to the length of the lesion, suggests the presence of a tumor in the wall of the colon. (On necropsy, the lesion proved to be a circumscribing adenocarcinoma of the descending colon with widespread metastases.)

Chloramphenicol and Dihydrostreptomycin (Chlorostrep) for Treatment of Diarrheas in Dogs and Cats

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IN RECENT YEARS, numerous antibiotics have been made available to the veterinary profession for treatment of enteric infections. Many of these drugs were at first used singly but later clinical studies have shown that antibiotic combinations often control the infection at a more rapid rate.

Antimicrobial synergisms have been found to exist when chloramphenicol (Chloromycetin) and dihydrostreptomycin are used in combination; their effect is enhanced rather than merely being additive.[1] Laboratory tests[2,3] and clinical observation[4] have shown that the antibacterial activity of chloramphenicol is augmented when combined with dihydrostreptomycin. Both antibiotics actually have a retarding effect on emergence of resistant organisms.[1] Both have been used separately in intestinal disorders[5-7] and have been suggested for the treatment of enteric infections of dogs.[8] Chloramphenicol was found[9] to be the most effective antibiotic *in vitro* against both gram-positive and gram-negative bacteria cultured from fecal material. It is readily absorbed from the intestinal tract,[10] while neither streptomycin nor dihydrostreptomycin are absorbed through the alimentary canal when taken orally.[11]

The present discussion covers the use of a combination of 125 mg. of chloramphenicol and 125 mg. of dihydrostreptomycin (Chlorostrep Kapseals*) as the sulfate for treatment of bacterial enteric infections of the diarrheal type, specifically those caused by Salmonella, Shigella, and other susceptible organisms found in small animals. This combined antibiotic has also been reported to be of value in the preoperative and postoperative treatment of many mixed bacterial infections encountered in bowel surgery in man.

METHODS

A total of 42 dogs and 1 cat were used to compare results with the combined antibiotic and results with our former treatment of bacterial and hemorrhagic enteritis of dog and cats.

Diagnosis of bacterial enteritis was made when signs of anorexia, vomiting, and profuse diarrhea were present. Hemorrhagic enteritis was diagnosed when bloody diarrhea with dehydration was also observed. All animals were examined for the presence of intestinal parasites.

TABLE I—Effect of S-R-D and Supportive Treatment on Bacterial Enteritis in 12 Dogs, All of Which Recovered. Treatment Was Continued Until Recovery

Dog No.	Breed	Age	Sex	Weight (lb.)	Duration of illness before treatment (days)	Improved (days)	Recovered (days)
1	French Poodle	7 yr.	female	12	1	3	5
2	Fox Terrier	13.5 yr.	male	32	1	3	5
3	English Bulldog	4 mo.	male	16	2	3	5
4	French Poodle	3 mo.	male	10	3	3	6
5	Dachshund	3 yr.	female	28	4	4	7
6	Cocker Spaniel	5 yr.	male	26	3	4	8
7	Toy Terrier	1 yr.	male	10	3	4	8
8	Cocker cross	4 yr.	female	24	2	3	5
9	Dachshund	7 yr.	female	25	3	4	8
10	Pomeranian	4 mo.	female	2½	2	4	7
11	Chihuahua	2 yr.	male	4	2	4	6
12	Cocker Spaniel	3 yr.	female	22	2	3	7

Twelve dogs suffering from bacterial enteritis were given the former treatment of S-R-D, a combination of kaolin and pectin (Kaopectate), and "triple sulfonamides" twice daily. Vitamin B complex was also given, in most instances, once daily intravenously. Animals showing dehydration received saline and glucose, both intravenously and subcutaneously, twice daily.

Drs. Schafer and Parker are small animal practitioners in North Hollywood, Calif.

*Parke, Davis and Company.

TABLE 2—Effect of a Combination of Chloramphenicol and Dihydrostreptomycin (Chlorostrep Kapseals) on Bacterial Enteritis in 20 Dogs, All of Which Recovered. Treatment Was Continued Until Recovery

Dog No.	Breed	Age	Sex	Weight (lb.)	Duration of illness before treatment (days)	Size and frequency of dosage*
1	Pekingese	4 yr.	male	15	2	1 twice daily for 2 days
2	Boxer	1 yr.	female	50	7	1 three times daily for 3 days
3	Cocker Spaniel	2 yr.	male	26	3	1 twice daily for 2 days
4	Wire-Haired Fox Terrier	2.5 yr.	female		3	1 twice daily for 4 days
5	Weimaraner	5 mo.	male	35	2	1 twice daily for 2 days
6	French Bulldog	1.5 yr.	male	25	2	1 twice daily for 3 days
7	Chihuahua	4 yr.	male	14	2	1 twice daily for 3 days
8	French Poodle	10 mo.	female	10	1	1 twice daily for 2 days
9	Great Dane	10 yr.	male	120	3	1 twice daily for 3 days
10	Cocker Spaniel	5 yr.	female	25	5	1 twice daily for 2 days
11	Cocker Spaniel	3 mo.	male	10	2	1 twice daily for 1 day
12	French Poodle	5 mo.	male	10	7	1 twice daily for 4 days
13	Pomeranian	3 yr.	male	....	2	1 twice daily for 3 days
14	Beagle	4 yr.	male	....	21	1 twice daily for 4 days
15	Dalmatian	5 yr.	male	....	3	1 three times daily for 3 days
16	Toy Poodle	3 mo.	male	....	2	1 twice daily for 2 days
17	Cocker Spaniel	1 yr.	female	....	3	1 twice daily for 3 days
18	Boxer	6 yr.	female	....	chronic	1 three times daily for 3 days
19	Boxer	3 yr.	male	....	30	1 three times daily for 7 days
20	French Poodle	1 yr.	female	....	3	1 twice daily for 2 days

*All dogs, except No. 9, were given one capsule initially.

The remaining 30 dogs and 1 cat received only the combined antibiotic; one capsule was administered twice daily to all animals, with the exception of dogs 2, 15, 18, and 19 (table 2). These dogs received one capsule three times daily.

RESULTS

When S-R-D and supportive therapy were used in treatment of bacterial enteritis, five to eight days' treatment (av. 6.4 days) was required for recovery (table 1).

Recovery followed in one to seven days with the combined antibiotic treatment (av. 3 days), even though dogs 18 and 19 (table 2) had chronic diarrhea for 30 days, or more, prior to treatment. Also, dog 5 was found to have a heavy infection of ascarids, which were removed after the diarrhea had stopped and dog 8 was treated for tapeworms two days after her stool became normal.

A Siamese cat had bloody diarrhea of two days' duration; dehydration was advanced and he was vomiting considerable bile. His temperature was 103.4 F. Supportive treatment consisted of the intravenous injection of a 5 per cent solution of dextrose and physiological sodium chloride. Therapy with one capsule of the combined antibiotic twice daily for six days was followed by recovery.

Dog 12 (table 2) had chorea of the right foreleg as well as clinical signs of a bacterial enteritis. Vitamin B complex was administered intravenously twice daily and 250 mg. of the combined antibiotic was given twice daily by mouth for five days. Following a relapse on the seventh day,

TABLE 3—Effect of a Combination of Chloramphenicol and Dihydrostreptomycin (Chlorostrep Kapseals) on Hemorrhagic Enteritis in 10 Dogs and 1 Cat, All of Which Recovered. Treatment Was Continued Until Recovery

Animal No.	Breed	Age	Sex	Weight (lb.)	Duration of illness before treatment (days)	Size and frequency of dosage*
1	Scottish Terrier	3 yr.	female	25	3	1 twice daily for 3 days
2	Cocker Spaniel	7 yr.	male	30	1	1 twice daily for 3 days
3	Pomeranian	11 yr.	male	10	1	1 twice daily for 2 days
4	Weimaraner	10 mo.	male	50	2	1 twice daily for 3 days
5	Dachshund	5 yr.	male	26	1	1 twice daily for 3 days
6	Cocker Spaniel	8 yr.	female	25	3	1 twice daily for 2 days
7	Cocker Spaniel	1.5 yr.	female	28	3	1 twice daily for 2 days
8	German Shepherd	8 mo.	male	35	2	1 twice daily for 3 days
9	Cocker Spaniel	2 yr.	male	....	7	1 twice daily for 4 days
10	Dachshund	2 yr.	female	....	5	1 twice daily for 4 days
11	Siamese cat	1 yr.	male	....	2	1 twice daily for 6 days

*All dogs, except No. 2 and No. 4, were given one capsule initially.

treatment was resumed twice daily, with complete recovery in four days. Weekly examinations for one month indicated that the chorea was also apparently brought under control.

Most patients had normal temperatures when treatment was started, but some had elevated temperatures.

It was apparent, when comparing the results with our former treatment, that a combination of chloramphenicol and dihydrostreptomycin gave more rapid recovery.

SUMMARY

A combination of chloramphenicol (125 mg.) and dihydrostreptomycin (125 mg.) (Chlorostrep), orally, gave more rapid recovery in the treatment of either bacterial or hemorrhagic enteritis of small animals than was observed when other medicaments were used.

One capsule of this combined antibiotic two or three times daily for one to seven days (av. 3 days) resulted in clinical recovery of all dogs. Six days' treatment was required for clinical recovery in a cat. There was no evidence of toxicity in any of the animals treated.

Animals exhibiting dehydration received supportive treatment in addition.

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Peritonitis in Goats Due to PPLO

A pleuropneumonia-like organism was recovered from a goat with fatal peritonitis. When cultures were injected intraperitoneally, they produced an acute fibrinous peritonitis in a sheep and a goat, and a chronic polyarthritis in another sheep; but a calf, 6 guinea pigs, and 6 mice were refractory. When injected intravenously, a sheep and a goat showed no ill effects. The organism was excreted in the nasal exudate of 1 goat and 1 sheep. Serum from the only goat examined contained complement-fixing antibodies. *In vitro,* the organism was highly sensitive to chloramphenicol and streptomycin, but resistant to penicillin.—*L. Laws in Austral. Vet. J. (Dec., 1956): 326-329.*

Anticoagulants and Cardiac Disease.—During a ten-year study, two groups of human patients with heart diseases (a total of 712 cases) were compared. The group which received anticoagulant therapy had a 14 per cent recurrence in five years while those not receiving the drug had 60 per cent. Death occurred during the second attack in 20.6 per cent of the treated group and in 53.0 per cent in the nontreated group. The chief contraindication for anticoagulants is the risk of hemorrhage but this appeared in only 3 per cent of the treated patients. It was controlled by injections of vitamin K.—*Sci. News Letter (April 27, 1957): 263.*

Epizootic Stringhalt in Horses.—In the fall of 1916, in New Zealand, about 100 horses in one area were affected with acute stringhalt and marked loss of condition. The pulse was usually fast, the temperature normal, but several had a tendency to "knuckle" at the fetlock, also a distinct roaring. Isolated cases have occurred since. Most of the horses were grazing but the condition was not due to a plant poison. Ten acutely affected animals died, the others recovered usually in six to 12 months. The cause has not been determined.—*New Zealand Vet. J. (March, 1956): 26.*

The Clinical Management of Snake Venom Poisoning in Domestic Animals

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THE INTENSE interest in animal venoms by research workers in various scientific disciplines is reflected by more than 4,100 references on this subject.[1] This literature contains many conflicting reports concerning the proper treatment of snake venom poisoning. For centuries, man has searched for a chemical substance which would neutralize venom once it has entered the body but, as yet, no simple, one-dose remedy has been found. There are few reports in the veterinary literature which consider the basic aspects of snake venom or the clinical management of venom intoxication in domestic animals. A recent survey in Florida showed that at least 360 domestic animals are treated there each year for snake bite.[2]

The authors estimate that thousands of animals are snake-bitten each year in the United States, especially in the southern and southwestern states. At least one species of poisonous snake has been reported to inhabit every state; therefore, it behooves the veterinary practitioner to know what snakes are found in his locality and how dangerous they are.

POISONOUS SNAKES OF THE UNITED STATES

The poisonous snakes which are native to the United States include the pit vipers (cottonmouth moccasins, copperhead moccasins, and 32 kinds of rattlesnakes) and the coral snakes. The large rattlesnakes of the genera Crotalus are the most dangerous snakes found in this country; the small pygmy rattlesnakes of the genera Sistrurus are much less dangerous. Few dogs or other small animals will survive the bite of a large eastern diamondback (*Crotalus adamanteus*) or western diamondback (*Crotalus atrox*) rattlesnake. Since less than 1 per cent of all human and animal bites are inflicted by coral snakes, this paper will consider only the pit vipers.

The pit vipers may be identified by a deep "pit" located between the eye and the nostril, also by their elliptical pupils, in contrast to the round pupil of nonpoisonous snakes (fig. 1). They have two well-developed fangs which are connected to venom sacs located on either side of the jaw. Rattlesnakes are recognized by the rattles attached to the tail; other pit vipers do not have rattles.

PATHOLOGY AND TOXICOLOGY

Snake venoms are complex chemical substances which are comprised chiefly of proteins.[3] The toxicity of snake venom has been attributed primarily to proteolytic enzymes, phosphatidases, and neurotoxins.[4] They also contain powerful "spreading factors," including hyaluronidase, which promote their dissemination into the body tissues. The proteolytic enzymes produce local tissue destruction and destroy the capillary vessels. Phosphatidases act on the heart and circulation and cause hemolysis of the red blood cells. Lysolecithin, formed in the tissues by venom, may also produce hemolysis. Histamine also is released when venom is injected into the body. The neurotoxins poison the nervous tissues and may cause motor and respiratory paralysis. The venom of North American pit vipers is primarily proteolytic and hematoxic, whereas the venom of coral snakes is primarily neurotoxic in action.

It has been shown[5] that snake venom is spread by the superficial lymphatic vessels and the tissue spaces. Although the fangs of a large rattlesnake may be over 1 inch in length, the venom is usually deposited in the subcutaneous tissue following a bite and only rarely as deep as the muscles. The pathological changes produced by pit viper venom include the local destruction of tissues and capillary endothelium, leakage of serum and blood into the tissues, and hemolysis of red blood cells which may produce anemia.[6] An animal may suffer from shock resulting from loss of blood and the

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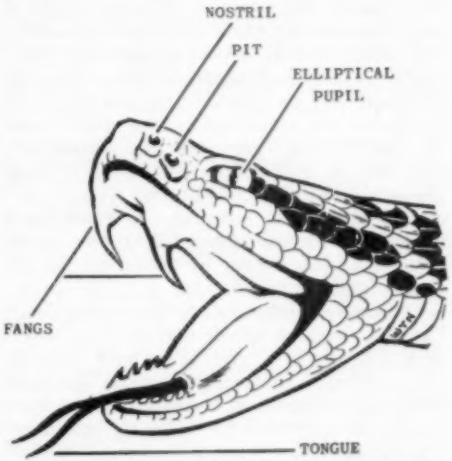
liberation of histamine. With severe envenomation, there may be involvement of the heart, kidneys, liver, brain, and intestinal mucosa. The coagulation mechanism may be altered so that the bleeding and coagulation times are prolonged. Gangrene, infection, and tissue slough are serious local complications. Snake bite wounds have been described[7] as contaminated, venom-laden, necrotic, and as anaerobic puncture wounds which provide an excellent medium for bacterial growth.

DIAGNOSIS

Poisonous snake bites in animals occur more frequently on the head, neck, shoulders, and forelegs. An animal may have been bitten several hours before it is found and brought in for treatment. There is usually evidence of one or two fang wounds (occasionally three or four if new fangs are growing in to replace old ones) surrounded by an area of edema. The wounds are painful when touched and, if the animal has been bitten on an extremity, it may limp. Frequently, a bitten animal has an enormously swollen head. Vomiting and diarrhea are common. An animal may exhibit excessive thirst and other signs of shock and, in severe envenomation, it may be unconscious, with or without convulsions. The edema and swelling are progressive if a large amount of venom was injected. Animals are usually listless and disinterested in eating for several days following snake bites. Minor bites generally produce minimal local swelling with no systemic signs, moderately severe bites produce extensive local swelling with few systemic signs, and severe bites produce extensive local swelling accompanied by the aforementioned systemic signs. Anemia and a local tissue slough almost invariably follow a severe envenomation.

TREATMENT

Treatment of poisonous snake bites is directed toward one or more of the following functions: (1) to prevent or retard venom absorption locally; (2) to remove the venom locally; (3) to neutralize the venom locally; (4) to neutralize the venom systemically; (5) to increase the elimination of the venom systemically (theoretical); (6) to prevent local complications; and (7) to prevent systemic complications. In evaluating drugs and surgical measures, it must be realized that many animals will survive minor bites without treatment; therefore, controlled therapeutic trials are of more value than isolated case reports. Also, some remedies such as potassium permanganate inactivate venom *in vitro* but are ineffective *in vivo*.



—Ross Allen's Reptile Institute, Silver Springs, Fla.

Fig. 1—The head of a pit viper showing diagnostic features.

Some of the factors which determine the severity of a poisonous snake bite are the size and species of the snake; the age, weight, and general condition of the animal; the location and nature of the bite (a glancing scratch as opposed to deep impregnation of the venom); and the toxicity of the venom of the specific snake. Envenomation is always more serious in small animals, as the ratio of units of venom injected to units of body weight is much greater. Bites on the head and trunk are more dangerous than bites on the extremities, for the venom is more rapidly absorbed into the body from these sites.

The present accepted therapy for snake bite includes a combination of medical and surgical measures. For convenience, the treatment may be divided into two phases: (1) first-aid treatment; and (2) hospital treatment.

First-Aid Treatment.—As soon as possible after an animal has been bitten, a tourniquet should be placed, when possible, several inches above the bite site. It should be applied only tight enough to occlude the spread of the venom through the superficial lymphatics and tissue spaces. It should

be released every ten to 15 minutes for a period of two or three minutes to restore circulation and it should be moved proximally, to keep just ahead of the advancing swelling. The purpose of the tourniquet is to impede the spread of venom until it can be mechanically removed or neutralized. The use of a tourniquet alone has been shown[8] to increase the survival time of bitten animals. In general, a tourniquet should not be used longer than two hours because of the danger of impairing the already damaged circulation.

Incision and suction should be employed locally to remove venom and its toxic products. The use of a tourniquet with incision and suction is referred[9] to as the "Jackson first aid treatment of snake bite." He demonstrated survival in dogs given four m.l.d.'s (minimum lethal doses) of venom and recommended cruciform incisions over the fang sites and in the surrounding edematous tissues. The incisions should be about ¼ inch long and from ⅛ to ¼ inch deep. Deep stab wounds into the muscles are unnecessary and may prove harmful. Multiple incisions may be necessary depending on the severity of the bite and the extension of the swelling but they increase the possibility of infection. We have found Cutter suction cups effective and easy to use for removing the venom. If several suction cups are available, they may be alternated every 15 minutes over the incisions. Suction should be carried out continuously and vigorously for two hours, then be maintained for 30 minutes out of an hour for an additional two or three hours, depending on the severity of the bite. During the interim, the wounds may be covered with saline compresses.

Other local measures which have been suggested are amputation, excision and suction, and refrigeration. Undoubtedly, amputation would prove effective but we do not recommend it; the treatment might be more harmful than the disease. Excision of an area 7 cm. in diameter surrounding the bite is an effective means of removing venom if the animal is seen within 60 minutes following the bite.[7] This form of treatment should be reserved for severe bites on the trunk when a large snake is involved. The "L-C method" (ligature and cryotherapy) has been recommended[10] for treating snake venom poisoning. This involves the use of a tight ligature, ice packs, and ethyl chloride.

Theoretically, freezing the tissues should lessen the enzymatic activity of the venom and retard its spread; however, it has not been proved a reliable form of treatment. As soon as the ice is removed, the venom spreads into the body and the frozen area is subject to necrotic degeneration and sloughing.[11] Also, treatment with ice does not seem practical in small animals. Therefore, it is our feeling that the "L-C method" should be rejected *in toto* until there is better evidence that it is not harmful and that it is beneficial.

Hospital Treatment.—Many drugs and chemicals have been tried for treating poisonous snake bites but none have shown specific value. However, some of the following are valuable adjuvant remedies; epinephrine hydrochloride, 3 to 5 minims of a 1:1,000 solution, is of value in treating the hypotension and shock which are invariably present in severe bites; intravenous saline, plasma, whole blood, and calcium gluconate may also be used for this purpose. Calcium gluconate solution may help prevent the hemolysis produced by pit viper venom. Meperidine hydrochloride (Demerol), 50 to 100 mg., may be given to alleviate pain.

The "three A's" (antivenin, antibiotics, and antitoxin) have been recommended,[12] in addition to surgical measures, for treating all serious snake bites. A new, potent product, Antivenin Crotalidae Polyvalent (North and South American Anti-snakebite Serum)*, is effective against the venom of North and South American pit vipers. One to five ampules (10 to 50 cc.) have been recommended, depending on the severity of the bite; 10 cc. should be infiltrated locally around the bite and the remainder should be given intramuscularly. A decision to use antivenin is usually determined by the severity of the bite, the size of the animal, and the desire of the owner to invest in this expensive biological product. In general, small animals require larger relative doses of antivenin. We have found a combination of penicillin and streptomycin effective in preventing bacterial infections secondary to snake bite but wide-spectrum antibiotics may be used if preferred. Enteric and coliform organisms have been reported[13] as the predominating bacteria in snakes' mouths and venom glands. Also tetanus and gas gangrene

*Wyeth Laboratories.

organisms have been reported in snake venom[14]; therefore, tetanus and gas gangrene antitoxin may be administered prophylactically.

There has been a recent tendency in medical and veterinary practice to rely on ACTH, cortisone, and antihistamine drugs as the primary treatment of poisonous snake bites; however, none of these drugs have been demonstrated experimentally to reduce mortality in dogs with venom poisoning.[15-17] Cortisone has been reported to prolong, but not save, life and it also apparently prevents sloughing at the bite site.[16] Antihistamines are contraindicated in treating animals with envenomation as these drugs appear to potentiate the toxic action of venom.[17]

SUMMARY

1) The four kinds of poisonous snakes found in the United States are listed, along with a discussion of the pathological and toxic effects produced by their venoms.

2) The use of a tourniquet and incision and suction are recommended as the first aid treatment of poisonous snake bites in animals.

3) In addition, the "three A's" (antivenin, antibiotics, and antitoxin) are suggested as the medical treatment.

4) Cortisone and ACTH may prolong life but have not affected the mortality rate.

5) Epinephrine hydrochloride, meperidine hydrochloride (Demerol), calcium gluconate, intravenous solutions, and blood transfusions may prove beneficial.

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Sulfonamide Poisoning in Mink.—A mortality of about 10 per cent is reported with three absorbable sulfonamides in different mink herds. Because of the high acidity of the urine, when on a high protein diet, the drug crystallizes readily in the kidneys. If it is necessary to use a medicated feed, antibiotics are preferred.—*A. Larson in Fur News (April, 1957): 18.*

Effect on Lice of Insecticides Given Systemically to Poultry.—Dieldrin was given orally, subcutaneously, or percutaneously (25 mg./kg. or less) to chickens. It had a powerful systemic effect on the body lice. The percutaneous route seemed most effective. The ova which hatched on the bird were rapidly killed. Mixing the insecticides with the feed might be a more economical method of treatment than dusting or spraying.—*Nature (April 13, 1957): 790.*

Somatotropin, the growth-promoting hormone of the pituitary gland, has been shown to affect cells directly. The addition of the growth hormone to cell cultures resulted in a consistent increase in the number of cells and in the rate of their protein synthesis, as compared with control tissue cultures.—*Science (April 5, 1957): 643.*

Nutrition

The Veterinary Practitioner and Nutrition

JAMES C. McCREA, D.V.M.

Platte City, Missouri

Efficient animal production is becoming more imperative every year, and a good sound feeding program is one of the many phases of livestock production that is going to keep your client and mine on the farm. In recent years, research workers have made remarkable discoveries in nutrition. The feed industry is making the benefits of these discoveries available in commercial feeds. Some drugs requiring federal sanction are included in these discoveries and they are made available in feed as soon as permission is obtained.

These discoveries should be of benefit to the livestock producers if used properly. Salesmen and feed dealers urge good nutrition but they do not, in most instances, understand such things as the "why" and the limitations of the products they sell. Until every feed store has a trained nutritionist on its staff, someone locally should be able to advise and explain the variable results and complications that will occur.

Many of our farm calls and office consultations are directly associated with nutrition. Some of these may involve rations for orphaned young or sick animals. Other problems may be concerned with deficiencies or overfeeding of ingredients such as proteins, vitamins, or minerals. A ration may be too bulky, too concentrated, or it may be nondigestible.

Nutritional requirements vary during the life cycle as well as within the species. The nutrients of livestock feeds vary with fertility of the soil, climatic conditions, seed varieties, and storage conditions. Specific enzymes in the feeds may destroy certain vitamins.

Research workers have discovered that extra vitamins, many amino acids, certain drugs, antibiotics, detergents, hormones, enzymes, rumen cultures, and unknown factors found in various substances aid, in one way or another, in the efficiency of the utilization of feed.

Nutrition is a complex and variable factor in itself and feed additives make it more so. Another highly variable factor in feeding livestock and poultry is animal husbandry. Good sound animal husbandry practices must be followed to get the maximum out of any feed. Many feeders feeding "growth stimulants" have never had their animals checked for external and internal parasites.

Keeping ourselves up-to-date on nutrition and all its additives can be done without sacrificing our practices. A few hours a week can accomplish this and the rewards will be gratifying. Here are a few suggestions:

1) Review your basic nutrition—get a new edition of Morrison's "Feeds and Feeding" (may be obtained from the Morrison Publishing Co., Ithaca, N.Y.).

2) Write the National Research Council, 2101 Constitution Ave., N.W., Washington 25, D.C., for "Recommended Nutrient Allowances for Domestic Animals"—poultry, swine, dairy cattle, beef cattle, sheep, and horses—($3.00 for all six copies or 50 cents each); and "Antibiotics in Agriculture" ($2.50).

3) Listen to farm radio programs and the items on feed manufacturers programs. This can be done while driving from farm to farm.

4) Read what the farmer reads so that you will know what he is talking about (he may have misunderstood the article). This includes farm magazines, newspapers, and advertisements by feed manufacturers.

5) At veterinary meetings, bring in speakers who are interested in nutrition.

6) Attend nutrition and feed conferences.

7) Read and ask for more articles on nutrition and feed medication in our veterinary journals.

8) Get experimental results on nutrition from land-grant colleges.

9) Know the analysis of feeds commonly sold in your area, especially medicated feeds (pick up analysis tags).

10) Know the price of the antibiotics and vitamins that go into a feed—what you can buy them for and what the feed manufacturer sells them for.

The U.S.D.A. is sharply curtailing aid to areas which have received drought relief in recent years. About half of the 600 counties getting feed grain, and two thirds of the 540 receiving hay and roughage, have been removed from the relief list.—*Cattleman (May, 1957): 21.*

Prepared by Dr. McCrea, Platte City, Mo., in cooperation with the American Association of Veterinary Nutritionists.

The Effect of Malnutrition on Viral Infection

Many observers have remarked that the runty pig is most apt to survive an attack of hog cholera. Also, those with much experience with the disease, since the introduction of improved management and feeding methods, may well question the earlier estimates that an average of as many as 3 per cent of the pigs in an infected, unvaccinated herd will survive; 100 per cent fatality seems much more common than 97 per cent. Both of these observations could well be correct, according to findings of virologists in recent years.

That unthriftiness could enhance resistance to an infectious disease is a thought diametrically opposed to most past teaching. It has been suspected in a few other diseases, such as blackleg, but the evidence can be clinically conclusive only in a highly infectious and highly fatal disease such as hog cholera.

This phenomenon is discussed in a recent article in the *Journal of Experimental Medicine* (Nov. 1, 1956: 687-706). The authors state that "The old concept that famine and pestilence go hand in hand does not always prove true." This had been noticed as early as 1789 when well-nourished children seemed the most susceptible to an infection. It had also been indicated in an experiment in 1942 when rabbits, under certain conditions of starvation, "became more resistant to infection with vaccinia." The consensus of virologists is said to be that "any influence inhibitory to host tissue metabolism will also be inhibitory to viral agents."

The article comments on the resistance shown by individuals already infected by some other disease. Such a resistance had been observed as early as 1840 with poliomyelitis, in 1928 with foot-and-mouth disease (aftosa), and experimentally in 1942 with swine influenza in mice.

It also had been shown that resistance to poliomyelitis was increased by a diet deficient in thiamine or in calories and that signs of the disease were delayed, but the eventual mortality not affected, by a depletion of certain protein constituents.

Hormone imbalance, degree of tissue hydration, environmental temperature, and age were other factors which influenced resistance to viral infections. Such conditions, because of their effect on the enzyme system and metabolism within the host's body cells, were believed to interfere with the multiplication of the virus in these cells.

Protein Use Affects Resistance

In recent experiments, using swine influenza virus in mice and Rous's sarcoma virus in chickens, the authors have shown that malnutrition resulted not in constant but in cyclic changes in resistance to viral infection. They found that with a low protein diet, there was an initial phase of increased susceptibility, a second phase of increased resistance, and a final phase of increased susceptibility.

They suggested that these phases were roughly correlated with the period of the utilization of depot fats (susceptibility), utilization of reserve protein (resistance), and the tissue breakdown due to protein starvation (susceptibility).

For an interesting review of factors involved in viral infection and in the development of immunity, *see* p. 505. This article mentions the enzyme system within the host cells upon which the virus particles are dependent for reproduction. It tells how simultaneous infection with two viruses can either enhance the pathogenicity of both (viral exaltation) or how one can prevent multiplication of the other (viral interference); also how resistance of body cells may be reduced following a subclinical infection (virus sensitization).

The fact that viruses reproduce within host cells explains why most of them are not affected by therapeutic agents. This may also indicate why immune antibodies, which seem to operate in body fluids, thus preventing systemic spread of the virus, can produce a solid immunity against a septicemic disease such as cholera and so little immunity against a localized disease such as the common cold.

These phenomena suggest new questions. For instance, what degree of nutritional change is required to produce significant cyclic phases in resistance? If it is minor, could it determine the severity of infection in an individual depending on which cyclic phase is present when the first wave of virus is released from the cells of the host? We still have much to learn about viral activities and resistance mechanisms.

Current Literature

ABSTRACTS

A New Feline Virus

During an attempt to grow *in vitro* the virus of panleukopenia (feline infectious enteritis), an unrelated agent producing intense cytopathogenic changes in kitten kidney epithelial cells was encountered. The origin of this agent was obscure, but could be consistently isolated from a single batch of panleukopenia virus-infected cat tissue. The KCD (kidney cell degenerating) virus grew well in most feline tissues when the latter were prepared either as suspended tissue fragment cultures or in the form of monolayers. It could not be maintained in suckling mice, adult mice, young rabbits, guinea pigs, or rats. Cytopathogenic changes were seen only in tissue cultures of cat origin. The chicken embryo and its surrounding extraembryonic tissues failed to support growth either *in ovo* or *in vitro*. The virus was serologically distinct from that of panleukopenia.

Infectivity for kittens was of a low order and could only be recognized by subsequent serological reactions. A limited survey of a local cat population showed neutralizing antibodies for KCD virus to be more prevalent among adult cats than in kittens of less than 8 weeks of age. In view of its low grade pathogenicity for the definitive host and its restricted species tropism *in vitro*, the KCD virus may, at the moment, be best described as an "orphan" virus of cats.—[*L. B. Fastier: A New Feline Virus Isolated in Tissue Culture. Am. J. Vet. Res., 18, (April, 1957): 382-389.*]

Characteristics of Colonial Forms of Vibrio Fetus

Stable, rough, smooth, and mucoid colonial variants were isolated from ten selected strains of *Vibrio fetus*, of bovine, ovine, and human origin. Colonies were stained with crystal violet and examined under a microscope, using indirect light.

Morphologically, all types were identical. The rough variants, only, were unstable in saline and acraflavine. Biochemically and antigenically, the mucoid variants were significantly different. All mucoid types were catalase negative, produced H_2S, and grew in stab cultures, whereas smooth and rough variants of the same strains were catalase negative, H_2S positive, and failed to grow in stab cultures. The two latter types had certain antigenic components in common but showed no cross-agglutination with homologous mucoid variants.—[*Jillian M. Bond: Characteristics of Colonial Forms of Vibrio Fetus. Am. J. Vet. Res., 18, (April, 1957): 449.*]

Enhancement of Listeriosis in Sheep

Blood, lymph node, and saliva from cattle with mucosal disease have been mixed with *Listeria monocytogenes* for exposure of test sheep to enhance the development of listeriosis. Blood and lymph node material from these epizootics of mucosal disease gave evidence of enhancing listeriosis. Material from two epizootics of mucosal disease failed to demonstrate the enhancing effect in a conclusive manner. The isolation of a second listeriosis-enhancing agent (similar to that previously reported, *Am. J. Vet. Res., 17,* (1956): 235-242) from another flock of sheep is described.

The enhancing effect of tissues containing a suspected agent has been obtained in 11 trials with 29 sheep of which 25 developed listeriosis and the organism was isolated from the central nervous system. For comparison, in 13 trials using 11 different materials, which were apparently devoid of enhancing effect, only 4 of 30 sheep developed listeriosis.

The listeriosis-enhancing factor in material from mucosal disease of cattle is probably different from that found in listeriosis in sheep.—[*C. Olson, Jr., O. D. Grace, D. Segre, and I. C. Blore: Enhancement of Listeriosis in Sheep with Material from Bovine Mucosal Disease. Am. J. Vet. Res., 18, (April, 1957): 303-309.*]

Blood Constituents in Ewes and Lambs

Values for blood cellular elements, hemoglobin, and serum levels of cholesterol, glucose, inorganic phosphorus, alkaline phosphatase, calcium, creatinine, protein-bound iodine, and nonprotein nitrogen were compiled for four years for Suffolk ewes and lambs maintained under laboratory conditions. Significant differences between lambs and mature ewes were observed in leukocytes, lymphocytes, neutrophils, erythrocytes, hemoglobin, inorganic phosphorus, and nonprotein nitrogen.—[*P. L. Hackett, D. W. Gaylor, and L. K. Bustad: Blood Constituents in Suffolk Ewes and Lambs. Am. J. Vet. Res., 18, (April, 1957): 338-341.*]

Hemagglutinating Properties of Salmonella Typhimurium

A bacterial hemagglutinin was demonstrated in 17 of 22 strains of *Salmonella typhimurium*. The hemagglutinin was heat labile and disappeared upon prolonged storage. Efforts to remove the principle by mechanical means were not successful. The results of this study indicate the necessity of carefully selecting strains free of hemagglutinating activity for the production of diagnostic antigens that are to be used with whole blood.—[*M. E. Harris and J. E. Williams: The Hemagglutinating Properties of Salmonella Typhimurium. Am. J. Vet. Res., 18, (April, 1957): 432-436.*]

FOREIGN ABSTRACTS

Diseases of Dairy Cows

The author briefly presents his substantiated views on the economical and public health significance of some diseases of dairy cattle. Essentially, the well-being and progress of the milk-production industry rests on the balance of three live forces: the producer or dairyman; the government, represented by its agencies in agriculture,

livestock and health departments; and the consumer. All three must know the conditions under which milk is produced and the means to fight diseases which are contagious to man or which can cause severe economic losses.

The diseases discussed are tuberculosis, brucellosis, Q fever, mastitis, anthrax, leptospirosis, nutritional disturbances, foot-and-mouth disease, infectious scours, parasites, and foot rot. The value of pasteurization and a sanitary code, and the responsibility of the dairyman in rural areas and in the public health are discussed.—[*J. Santivanez M.: Economical and Public Health Significance of Some Diseases of Milk Cattle. Ciencias Vet. (Mexico), 1, (Nov.-Dec., 1956): 193-204.*]—O. A. LOPEZ-PACHECO.

BOOKS AND REPORTS

Venoms

This book is a compilation of the papers presented at the First International Conference on Venoms at the annual meeting of the American Association for the Advancement of Science, Berkeley, Calif., Dec. 27-30, 1954.

The field is covered from protozoa to mammals in both land and sea life. The physiological properties of venoms, the chemistry, pharmacological action, signs, and treatment are well discussed. Extensive references are given.

It would seem that in the Asian countries, where snake bite is more common than in any other part of the world, they do not use incision and suction but depend upon antivenin, with good results. In Bangkok, where around 2,000 cases of snake bite are treated each year, one speaker reported that all patients brought alive to the Institute of the Thai Red Cross recovered when treated with antivenin alone. In the western countries, incision and suction are recommended and antivenin is used with caution even after sensitivity tests are given. These statements might lead one to wonder if the dark-skinned races are not less sensitive than those with lighter complexions.—[*Venoms. Edited by Eleanor E. Buckley and Nador Porges. 467 pages. Publication of the American Association for the Advancement of Science, Washington, D. C. 1956. Price not given.*]—G. W. McNUTT

Applied Animal Nutrition

This book is concise, although relatively comprehensive. One of its objectives is to bring closer together the theory of nutrition and the practice of animal feeding. The author drew directly on his extensive knowledge and wide experience of the subject. References have been omitted except to mention a limited number as suggested additional reading.

The sections into which the subject matter is divided concern (1) feedstuffs, (2) nutritional requirements of animals, (3) nutritional characteristics of feeds, (4) formulation of rations, and (5) appendices covering feeding guides, controls, tables, and charts. Sections have introductions and summaries and one is tempted, at times, to read both of these prior to the body of the section in question because of the helpful orientation provided. Figures and graphs are frequently used to supplement the text. Helpful explanatory discussions are included of terms used in nutrition of livestock such as "T.D.N." and "biological value."

Inclusion of antibiotics, along with minerals and vitamins, in the section on requirements of livestock is an indication of the changes that have occurred in recent years in the field of livestock nutrition. Helpful "thumb rules" for feeding various classes and ages of livestock are provided. Of necessity, these are brief and this, at times, makes it difficult to vary the emphasis desired on different details. An example is the reference to providing hay to calves at 2 weeks of age on one page and at 1 week on another. As no reasons are given for withholding roughage, one wonders why not merely provide hay from the beginning in all instances. Regardless, it should prove to be a useful book for any veterinarian who engages in large animal work.—[*Applied Animal Nutrition. By E. W. Crampton. 478 pages. Illustrated (28 figures, 86 tables, 15 charts). W. H. Freeman and Co., 660 Market St., San Francisco, Calif. Price $6.50.*]—W. D. POUNDEN.

Cryptococcosis (Torulosis or European Blastomycosis)

The authors have attempted, successfully, to present the sum of pertinent information on cryptococcosis as a disease of man and other animals. The role of *Cryptococcus neoformans* as a mycological entity and its host relations as a disease agent are lucidly portrayed by means of a comprehensive pictorial and textual treatment. The problem is clearly defined in a brief introduction. In the following nine chapters, the authors describe and illustrate the wide range of clinical and pathological manifestations produced in man and other animals by the fungus *C. neoformans* and, at the same time, provide historical, epidemiological, therapeutic, and technical information for those interested primarily in these aspects of the subject. An appendix of the most important mycological and histopathological techniques has been included as an aid to laboratory diagnosis. In the bibliography, there are 504 references, including reports of spontaneous cryptococcosis in animals as late as 1955.

There are 82 figures in black and white; most are of several parts and many occupy a full page. In general, they are informative, well selected, and adequately reproduced. The four one-page color plates are beautiful and informative. The text is lucid and concise. The paper, printing, and binding are of good quality.

This book is the outcome of the joint efforts of a medical mycologist and a pathologist and reflects credit to both. The authors indicate that this is the first of a series of monographs on individual systemic mycoses. It is hoped that subsequent numbers

of the series will be of comparable over-all quality and not long delayed. This book should be of general interest to any veterinarian for both he and his patient may be directly concerned. It is especially recommended to veterinary pathologists and mycologists and to veterinary diagnostic laboratories and clinicians in our veterinary schools.—[*Cryptococcosis (Torulosis or European Blastomycosis). By M. L. Littman and Lorenz E. Zimmerman. 205 pages. Grune and Stratton, Inc., 381 Fourth Ave., New York 16, N. Y. 1956. Price $8.50.*]—CHARLIE N. BARRON.

Infectious Diseases of Swine

This is an excellent review of the literature concerning the more important infectious diseases of swine in North America. It is a valuable contribution to an area of veterinary medicine which has developed largely within the span of the author's extensive experience as a laboratory diagnostician, and frequently a field consultant, in the region with the world's greatest concentration of swine.

The review deals primarily with the history and means of controlling these swine diseases; their etiology and pathology are, in most cases, only incidentally mentioned. The development of knowledge about each disease is interestingly chronicled, although the reader who is not familiar with swine diseases may be confused by some apparently contradictory comments.

For instance, an early observer found "sulfonamides" of no value in "enteritis" while several later observers reported them of great value in certain forms of enteritis (the third paragraph under a carelessly placed subheading on page 36, seems to include transmissible gastroenteritis as also being benefited; however, the statement probably does not refer to that disease).

This good treatise could have been clarified in this and several other places and generally improved had it been well edited. Also, considering the author's experience, it might well have included more of his own observations and conclusions. Nevertheless, what is reported is as accurate and up-to-date as possible in this rapidly changing field.—[*Infectious Diseases of Swine. By H. C. Smith. 96 pages. Illustrated. Veterinary Medicine Publishing Co., 803 Livestock Exchange Bldg., Kansas City 2, Mo. Price not given.*]—W. A. AITKEN.

Veterinary Clinical Diagnosis

The Austrian editions of this text are based on notes prepared by the author for student use many years ago. These notes were repeatedly rewritten and expanded as a result of experience gained through years of teaching and examining. The result is a book of convenient size with well-chosen content, logically presented on a systematic basis. Attention is given to every essential detail.

The translator has made little change in those chapters dealing with the general clinical examination of the patient. The original manuscript was based mainly upon the examination of the horse, small animals, and the fowl. The English edition is more than a mere translation, as much material has been added that has to do with diagnostic procedures in the ox, sheep, and pig. This has necessitated the almost complete rewriting of some chapters to have them meet the needs of British students.

A valuable factor in Dr. Wirth's text is the abundance of helpful illustrations. This feature is not only continued in the English edition but its value is enhanced by the addition of 92 new photographs.

In the preface to the first edition, Dr. Wirth makes the following statement: "This book is intended to provide an introduction to clinical examination and diagnosis for the student entering the clinic for the first time. As a general understanding of the whole of this important subject should be the first consideration, the book has been kept short, any idea of an exhaustive treatment of the subject being renounced."

The American student will find this book adequate for the purpose for which it is intended.—[*Veterinary Clinical Diagnosis. By Prof. Dr. David Wirth. 232 pages. First English translation, Bailliere, Tindall and Cox, 7 and 8 Henrietta St., London, W. C. 2, England. 1956. Price $6.00.*]— C. H. COVAULT.

Horse Psychology

Despite its importance to horsemen and the fact that observations on the matter have been set down for hundreds of years, the psychology of the horse is relatively unexplored. This is because the horse is an expensive subject for controlled laboratory study, horse behavior varies as widely as human behavior, and is affected by human psychology since the subject animal can not well be separated from his master.

In the chapter on vision, the author opines that the horse, like other mammals except man, the ape, and monkeys, can not distinguish colors except in a primitive and rudimentary way. Whether horses have better night vision than man is doubtful, the author says. But, although the horse has a small field of binocular vision, it can see toward the rear because its eyes are set on the side of the head.

The author seems to think there is some relationship between coat color and temperament — blacks being touchy; chestnuts (like redheads), hot tempered; greys, docile and dependable; and bays, "self-willed and mulish."

As for intelligence, there is no definite conclusion except the indisputable one that some horses are more intelligent than others. Horses can be trained to do a number of things that seem to reflect intelligence, but it may be that such demonstrations are a matter of "cueing" by the trainer.—[*Horse Psychology. By Moyra Williams. 191 pages. Illustrated with drawings. Countryman Press, Woodstock, Vt. 1957. Price $4.75.*] *(As reviewed by J. A. Estes in* Blood Horse, *March 23, 1957: 794-795.)*

THE NEWS

Symposium on Reproduction and Infertility

The Third Symposium on Reproduction and Infertility will be held at Colorado State University, July 1-3, 1957. The program follows:

MORNING SESSION—Monday, July 1

New Aspects of Vibriosis in Sheep—Rue Jensen and V. A. Miller, Colorado State University, Fort Collins.
New Aspects of Vibriosis in Cattle—Kenneth McEntee, New York State Veterinary College, Ithaca.
Catarrhal Vaginitis of Cattle—D. G. McKercher, University of California, Davis.
Epididymitis of Rams—Blaine McGowan, University of California, Davis.

AFTERNOON SESSION

Neoplasms of Bovine Genitalia—Wayne A. Anderson and C. L. Davis, Animal Disease and Parasite Research Division, U.S.D.A., Denver.
Discussion panel—Rue Jensen, chairman; V. A. Miller, Blaine McGowan, D. G. McKercher, Kenneth McEntee, C. L. Davis; and Malcolm S. Trueblood, University of Wyoming, Laramie.

MORNING SESSION—Tuesday, July 2

Recent Studies on the Mechanism of Ovulation in the Cow—William Hansel, Cornell University, Ithaca.
Motion picture—Ovulation in the Rat—Richard J. Blandau, University of Washington, Seattle.
The Effects of Administration of Gonadotropins on the Maturation of the Egg Nucleus in Ewes—R. O. Berry, A. & M. College of Texas, College Station.
Relation of the Nervous System to Implantation—A. V. Nalbandov, University of Illinois, Urbana.
Cytological Changes in the Bovine Corpus Luteum During Early Pregnancy—R. C. Foley and J. S. Greenstein, University of Massachusetts, Amherst.
Ovarian Function, Blood Biochemistry, and Reproductive Performance of Repeat Breeder Cows—James A. Williams, Clifford W. Duncan, and Donald H. McWade, Michigan State University, East Lansing.

AFTERNOON SESSION

Biosynthesis of Steroid Hormones—L. T. Samuels, University of Utah, Salt Lake City.
Determination of Corticosteroids in Cattle—William G. Robertson, Schering Corp., Bloomfield, N. J.
Maintenance of Human Pregnancy with Progestational Compounds—E. C. Reifenstein, Jr., E. R. Squibb and Sons, New York, N. Y.
Estrogen Determination in Blood and Body Fluids of Cattle—Joel Bitman, T. R. Wrenn, and J. F. Sykes, Dairy Husbandry Research Branch, U.S.D.A., Beltsville, M.D.
Estrogenic Steroids in Pregnant Swine Urine—H. E. Bredeck and D. T. Mayer, University of Missouri, Columbia.
Dinner—Problem of Low Level World-Wide Radioactive Fall-Out—Wright H. Langham, Los Alamos, N. M.

MORNING SESSION—Wednesday, July 3

Controlled Estrus in Cattle—W. E. Petersen, James R. Nichols, Edward Graham, and John Donker, University of Minnesota, St. Paul.
Discussion panel—F. X. Gassner, Colorado State University, chairman; W. E. Petersen, Edward Graham, John Donker; John E. Nellor, Michigan State University, East Lansing; and S. W. Mead, University of California, Davis.

Brigadier General Wayne O. Kester (left), AVMA president, Dave Garroway, and Dr. J. Lavere Davidson of the Upjohn Company, recently appeared on Garroway's NBC-TV "Today" show, which was devoted to the advances in veterinary medicine.

Problems of Infertility in Dairy Herds—Phillip M. Hinze, Carnation, Wash.
Present Status of Therapy in Animal Infertility—H. J. Hill, Colorado State University, Fort Collins.
Male Infertility—Henryk Nowakowski, Medical University Clinic, Hamburg-Eppendorf, Germany.

AFTERNOON SESSION

Isotopic Studies of Semen Metabolism—R. J. Flipse, Pennsylvania State University, University Park.
Round-table discussion on artificial insemination—Frozen Semen—H. J. Hill, chairman; S. W. Mead; Richard Kellogg, Columbus, Ohio; M. H. Ehlers, State College of Washington, Pullman; and Charles P. Stroble, University of Wyoming, Laramie.

Committee of Veterinary Toxicologists Organized

A meeting of veterinarians held at Logan, Utah, on April 17, 1957, resulted in the formation of an organization to be known as the Committee of Veterinary Toxicologists.

The group met to discuss various problems dealing with chemical toxicosis of animals and particularly the problem of fluorosis.

The office of the committee will be 1102 E. Main St., Puyallup, Wash., with Dr. William F. Harris, of the same address, as secretary.

Training Program for Steroid Biochemistry

Applications are now being accepted for the second course in the training program for steroid biochemistry to start Oct. 1, 1957. The program is conducted through the cooperative efforts of the Worcester Foundation for Experimental Biology; the Department of Chemistry, Clark University, Worcester, Mass.; and the Department of Biochemistry, College of Medicine, University of Utah, Salt Lake City.

The training program is sponsored by the National Cancer Institute, National Institutes of Health, U.S.P.H.S., to provide specialized training for individuals interested in steroid investi-

gation. Two groups of candidates will be selected and will receive stipends during the period of training.

The closing date for applications will be Aug. 15, 1957. Requests for applications and additional information should be sent to Dr. Kristen Eik-Nes, Department of Biochemistry, College of Medicine, University of Utah, Salt Lake City; or to Dr. Frank Ungar, Department of Chemistry, Clark University, Worcester, Mass.

Course in Diagnostic Methods in Veterinary Mycology

During the week of Feb. 25-March 1, 1957, the Communicable Disease Center of the U. S. Public Health Service, presented, at Chamblee, Ga., a course in laboratory diagnostic methods in veterinary mycology. This course was designed to familiarize veterinarians, laboratory directors, and others concerned with procedures for detecting, isolating, and identifying the fungi which produce mycotic infections in lower animals. The etiological agents were studied in tissue and in culture. Methods of diagnosis covering specimen collecting, culture mediums, laboratory animal inoculations, biochemical, and other procedures were taught, used, and demonstrated. The symptomatology, epidemiology, and public health implications of the animal mycoses were also stressed.

Nineteen students attended this course.

The next course will be held at the Communicable Disease Center, February, 1958. Application for the next course can be made after July 1, 1957, and should be addressed to the Communicable Disease Center at Chamblee, Ga.,

s/ROBERT D. COURTER, *Assistant Chief, Veterinary Public Health Section.*

AMONG THE STATES AND PROVINCES

District of Columbia

Veterinary Academy Organized.—The District of Columbia Academy of Veterinary Medicine has been organized by a group of greater Washington veterinarians, to further knowledge of small animal medicine and surgery.

Postgraduate courses will be offered by the Academy in the latest surgical and diagnostic techniques.

Membership is open to licensed veterinarians actively engaged in care and treatment of small animals, who are also members of the AVMA.

The new officers are: Robert M. Lynn, president; W. Harry Cowan, vice-president; Jean S. Goudy, secretary; Louis Sordo, treasurer; and H. Carlton Newman, president-elect.

On April 4, 1957, Dr. Gerry B. Schnelle of Angell Memorial Animal Hospital, Boston, discussed "X-ray Diagnoses, with Reference to Intervertebral Disc Protrusion Syndrome and Congenital Hip Dysplasia." Eighty veterinarians were in attendance.

Dr. Robert M. Lynn

s/MARSHALL J. WAPLE, JR., *Publicity Chairman.*

Colorado

Dr. Adams Awarded Fulbright Scholarship.—Dr. O. R. Adams, professor and head, Department of Veterinary Clinics and Surgery, Colorado State University, has been awarded a Fulbright scholarship to Kabete, Kenya, East Africa, starting in July, 1957. Dr. Adams will aid a new veterinary school there in organizing large animal and ambulatory clinics. He will also do part-time research in the virus diseases occurring in that area. This veterinary school provides African students a six-year course and grants them a certificate, but not a degree of doctor of veterinary medicine.

During his travels, Dr. Adams hopes to visit some veterinary schools in Europe and the research station at Onderstepoort, Union of South Africa.

Idaho

Southwest Idaho V.M.A.—The Southwest Idaho V.M.A. met at the Homestead Inn in Nampa for its regular dinner meeting on March 14, 1957. The following reports were given: Mr. Costello Holmes (AVMA insurance program); A. P. Schneider, state veterinarian (legislation affecting the livestock industry); John Williams (resumé of papers presented at the Salt Lake City meeting); V. Pennell (report on recent Colorado veterinary meeting); and F. Fisher, Idaho veterinarian in charge, ARS (diagnosis of scabies in livestock). Dr. M. Ickes, president, moderated the discussions following presentation of each report.

s/MICHAEL INVERSO, *Correspondent.*

Illinois

Northern Illinois V.M.A.—The thirty-sixth semiannual meeting of the Northern Illinois V.M.A. was held April 17, 1957, at the Faust Hotel, Rockford, with 136 in attendance.

The program consisted of the following speakers and their topics: F. E. Connor, Morris (dwarfism in cattle); P. T. Candlin, Madison, Wis. (trimar anesthesia in the cat, and resuscitation in the dog); A. K. Merriman, Springfield (recent progress of state legislative measures); P. T. Gambrel, Winnebago (remarks on I.S.V.-M.A. committee work); G. T. Woods, Urbana (relationship of practicing and extension veterinarians); Douglas Hansen, Chicago (general work of the pure food and drug administration); W. H. Beckenhauer, Lincoln, Neb. (swine diseases); and Harold Held, Freeport (dairy cattle practice).

It was voted to donate $100 to the AVMA Research Fund; and also to sponsor a one-day clinic at the St. Charles Livestock Pavillion, tentatively scheduled for May 15, 1957, under the chairmanship of Dr. H. P. Wessels, Geneva.

The program was followed by a cocktail hour. The fall meeting of the Association has been scheduled for Wednesday, Sept. 18, 1957, at the Faust Hotel, Rockford.

s/JAMES G. HARDENBERGH, *Secretary.*

Iowa

North Central Iowa V.M.A.—The annual meeting of the North Central Iowa V.M.A. was held Thursday, April 18, 1957, at the Warden Hotel, Fort Dodge, with 101 in attendance.

The following addressed the group: A. W. Deem, Colorado State University, Fort Collins; A. A. Case, University of Missouri, Columbia; Bernard Ebbing, Rath Packing Co., Waterloo; and R. T. McCarty, Wyeth Laboratories, Inc., Alvarado, Texas.

Officers for the coming year are: Wayne Emerson, Eagle Grove, president; John Morton, Webster City, president-elect; and H. Englebrecht, Fort Dodge, secretary-treasurer.

s/H. ENGLEBRECHT, *Secretary.*

New Hampshire

State Board Examination.—The New Hampshire State Board of Veterinary Examiners will hold examinations Friday, July 5, 1957, at 9:00 a.m. in Concord. Applications must be on file with the secretary no later than June 20. Dr. J. P. Seraichick, Chesterfield Rd., Keene, N. H., secretary.

FOREIGN NEWS

Argentina

Veterinary Association to Have New Quarters.—The Sociedad de Medicina Veterinaria, Buenos Aires, founded in 1897, has started the building of its new premises which will cover nearly 285 square meters; the plan includes a library, a spacious salon for both scientific and cultural sessions, bar and lounge, besides the offices for the permanent staff and offices for both secretary and president.

s/HECTOR G. ARAMBURU, *Prosecretario.*

STATE BOARD EXAMINATIONS

KENTUCKY—June 17 and July 29, 1957, Frankfort. J. K. Bushnell, Paris, Ky., secretary.

MARYLAND—June 19, 1957, Baltimore. Dr. Harold S. Gober, 5400 Park Heights Ave., Baltimore 15, Md., secretary.

MINNESOTA—July 8-9, 1957, St. Paul. A. C. Spannaus, executive secretary, Minnesota Veterinary Examining Board, Waconia, Minn.

MONTANA—June 24-26, 1957, Bozeman. J. W. Safford, secretary-treasurer, Montana Board of Veterinary Medical Examiners, Capitol Station, Helena.

NEW JERSEY—June 28-29, 1957, Trenton. Joseph A. S. Millar, P.O. Box 172, Deal, secretary.

NEW YORK—June 12-13, 1957. Practical examination, Ithaca. Mr. James O. Hoyle, secretary, 23 S. Pearl St., Albany. Week of July 8, 1957. Written examinations: New York City, Albany, Syracuse, Buffalo, Rochester.

NORTH CAROLINA—June 24-26, 1957. Asheville. Dr. James I. Cornwell, secretary, 65 Beverly Road, Beverly Hills, Asheville.

SOUTH DAKOTA—June 24-25, 1957. Pierre. Glenn B. Rea, Livestock Sanitary Board, State Office Building, Pierre, S. Dak., secretary.

TENNESSEE—June 24-25, 1957. Nashville. Dr. W. O. Greene, secretary, State Office Bldg., Nashville.

NEW HAMPSHIRE—July 5, 1957, Concord. J. P. Seraichick, Chesterfield Rd., Keene, N. H., secretary.

DEATHS

Star indicates member of AVMA

★**Alvin H. Dowdy** (OKL '51), 31, Duncan, Okla., died April 3, 1957, from injuries received in an automobile accident. Dr. Dowdy, a general practitioner, was a member of the AVMA.

O. W. Holman (STJ '14), 84, Frankford, Mo., died March 18, 1957. Dr. Holman is survived by a daughter, five grandchildren, and three great grandchildren.

Edgar L. Houck (MCK '13), 68, died in Winnipeg, Man., on Feb. 2, 1957.

Dr. Houck was a past-president and director of the Manitoba Veterinary Association and had been a member of the AVMA.

He is survived by a daughter.

★**Edwin P. Johnson** (MSU '25), 62, died in Roanoke, Va., on March 21, 1957.

Dr. Johnson had been a member of the staff at Virginia Polytechnic Institute, Blacksburg, Va., since 1930. He had obtained his Ph.D. degree of the University of Virginia in 1935.

Dr. Johnson was one of the leading research investigators in the field of poultry diseases and was credited with the development of a vaccine against Newcastle disease. He served as a member of the Inter-regional Advisory Com-

mittee on Newcastle Disease and Other Respiratory Diseases of Poultry and was the author of a number of scientific articles on this subject, the latest appearing in the December, 1954, JOURNAL.

He had served as AVMA representative to the National Research Council (Division of Biology and Agriculture) for a number of years. Dr. Johnson was a former president of the Virginia V.M.A. and had been a member of the AVMA since 1927.

Fred J. Lambeck (GR '12), 75, Kalamazoo, Mich., died March 26, 1957. Dr. Lambeck was a general practitioner. His widow, a son, and a daughter survive.

Arnold F. Lees (ONT '89), 90, Red Wing, Minn., died March 18, 1957. Dr. Lees was a member of the State Veterinary Examining Board and had served as Minnesota's first state veterinarian. His widow, two sons, a daughter, and eight grandchildren survive.

★**August C. Luh** (KCV '17), 81, De Soto, Mo., died April 7, 1957. Dr. Luh had practiced in Victoria, Mo., for many years. He was a member of the Missouri Veterinary Medical Associaton and of the AVMA to which he was granted life membership in 1955. His survivors include his widow, a daughter, and two sons.

W. M. McLeod (ISC '17), 65, Manhattan, Kan., died May 6, 1957. Dr. McLeod had been a member of the faculty at the School of Veterinary Medicine, Kansas State College, since 1919, and was head of the Department of Anatomy at the time of his death. He was a member of the Kansas State Veterinary Medical Association and had been a member of the AVMA. He was also a member of Phi Kappa Phi, Sigma Xi, and Gamma Sigma Delta.

Dr. McLeod is survived by his widow, three daughters, and six grandchildren.

★**Harry K. Miller** (NYC '91), 92, Mamaroneck, N. Y., died April 8, 1957, following a brief illness.

Born at Manheim, Pa., Oct. 13, 1864, Dr. Miller was a pioneer in small animal practice, having established one of the first small animal hospitals in the country shortly after his graduation from veterinary college. After moving to Mamaroneck in 1902, he developed the veterinary practice now known as Drs. Miller and Clark.

He was a former president of the New York State and New York City Veterinary Medical Associations and was an honorary member of the New York City and West Chester County Associations at the time of his death.

Dr. Miller joined the AVMA in 1913 and had been in continuous good standing since then. He was also active in civic, church, and service club affairs.

He is survived by a son, Dr. Walter R. Miller (COR '26), of Mamaroneck, a daughter, two brothers, two sisters, and five grandchildren.

Edwin L. Miller (ONT '18), 65, died in Houlton, Maine, on Jan. 16, 1957.

Dr. Miller was fatally injured in an automobile accident while returning to his home from the winter meeting of the Maine V.M.A.

Dr. Miller held memberships in the National Association of Federal Veterinarians, the New England V.M.A., the Vermont V.M.A., and the Maine V.M.A. He had been a member of the AVMA.

He is survived by a daughter.

★**Roy V. Rafnel** (CIN '11), 70, Tallahassee, Fla., died on April 2, 1957, after a short illness. Dr. Rafnel was director of the Meat Inspection Division, Livestock Sanitary Board, Florida Department of Agriculture. He had lived in Tallahassee for the past seven years.

Dr. Rafnel served in varying capacities for various state and federal agencies prior to his retirement from federal meat inspection service about seven years ago. At that time, he was inspector in charge of meat inspection in St. Louis, Mo. Prior to going to St. Louis, Dr. Rafnel had served in the Washington office for several years.

He is survived by his widow and a daughter. Dr. Rafnel was a member of the AVMA for many years.

Col. James R. Shand (CVC '07), 76, retired Army Veterinary Corps officer, died on April 11, 1957, in Palo Alto, Calif., where he had made his home since 1936.

Born in Indiana in 1890, he was appointed a contract quartermaster veterinarian in the Army shortly after graduation from Chicago Veterinary College. He had seen service in various parts of the world including, first, the Philippines from 1907 to 1914, then with the U. S. Forces in China at Tientsin and at Peking where he was supervising veterinarian for the American legation.

When the Army Veterinary Corps was established in 1917, he was commissioned first lieutenant and sent to Camp Dix. He was promoted to captain in 1920, to major in 1921, to lieutenant colonel in 1927, and to colonel in 1934. In 1927, he was again sent to the Philippines. He returned to the States and served at Fort Lewis, Washington, from 1930 to 1935. After spending some time in Fitzsimmons General Hospital, Col. Shand retired because of disability incurred in line of duty in 1936 and came to Palo Alto to live. He had been ill for about two months prior to his death.

Surviving are his widow, the former Louise O. Schwender, whom he married in 1921; a brother, a sister, and a nephew.

Colonel Shand joined the AVMA in 1913; he was a Mason and a Shriner. Interment was at Golden Gate National Cemetery.

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Cleveland—August 19-22

Ohio Vacation Opportunities

With the 1957 convention city located in north central Ohio on the southern shores of Lake Erie, vacation opportunities in the state should appeal to veterinarians and their families from all parts of the country.

Attractions for visitors to Cleveland include the Municipal Stadium where the Cleveland Indians play baseball; Severance Hall for the Cleveland Symphony; Terminal Tower, Nela Park, Cultural Gardens, the Cleveland Art Museum, and an outstanding Health Museum. West of Cleveland, midway between the convention city and Toledo, is Sandusky, the "jumping off" place for a cluster of scenic islands set aside for recreation and the fruit orchards of the Marblehead Peninsula. A ferryboat connects the islands and Cleveland, 50 miles away. Fishing, camping, swimming, and scenery, including several interesting caves, await vacationers.

The Ohio River valley is packed with caves in the bluffs flanking sections of the river. These caves served as bases for pirate raids on river shipping in the early days.

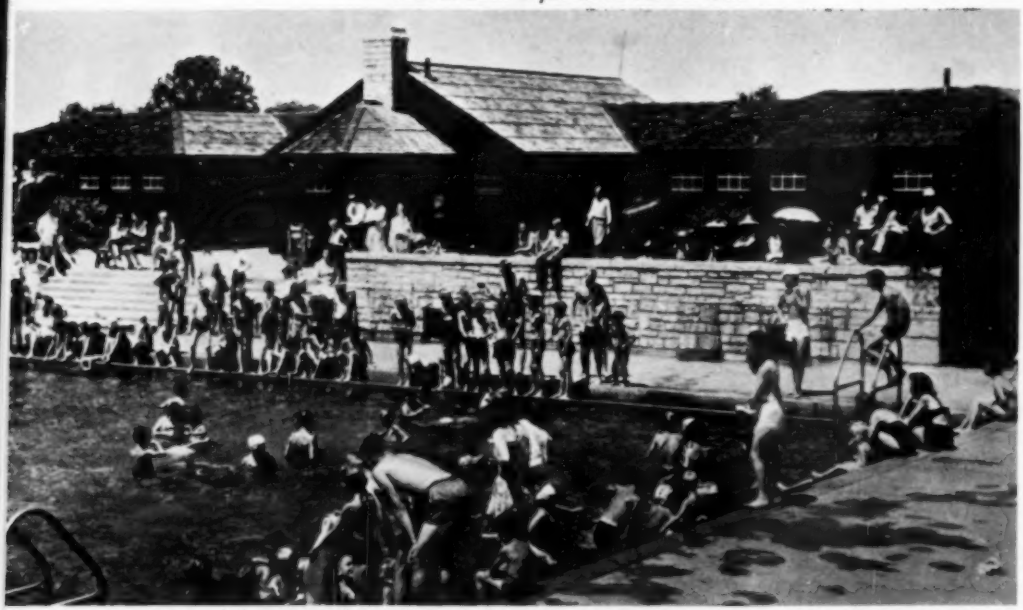
Prehistoric Ohio was a home to the mound builders and the most famous, Serpent Mound, is located in Adams County, off route 23, in southern Ohio. All told, the mound builders created over 10,000 monuments of earth in Ohio.

General Anthony Wayne, "mad Anthony" of Revolutionary War fame, conducted several campaigns against Ohio Indian tribes when he was chief of the Army under President George Washington. His winter quarters in western Ohio, Fort Recovery, have been reconstructed near U. S. highway 127.

There are 54 park areas throughout Ohio, all but one located close to state roads or U. S. highways. Most of the parks have picnicking areas; 25 have special swimming facilities and nine permit swimming although no guards or piers are provided. The lakes at Yellow Springs and Laurelville require reservations and permit swimming in groups. Forty-four of the parks permit fishing, with an Ohio fish and game license, of course. Forty of them permit boating and 25 offer boats to rent.

Four of the state parks have vacation cabins.

Pool at John Bryan State Park in Ohio



Many of the state parks in Ohio provide supervised areas for swimming, vacation cabins, and restaurants.

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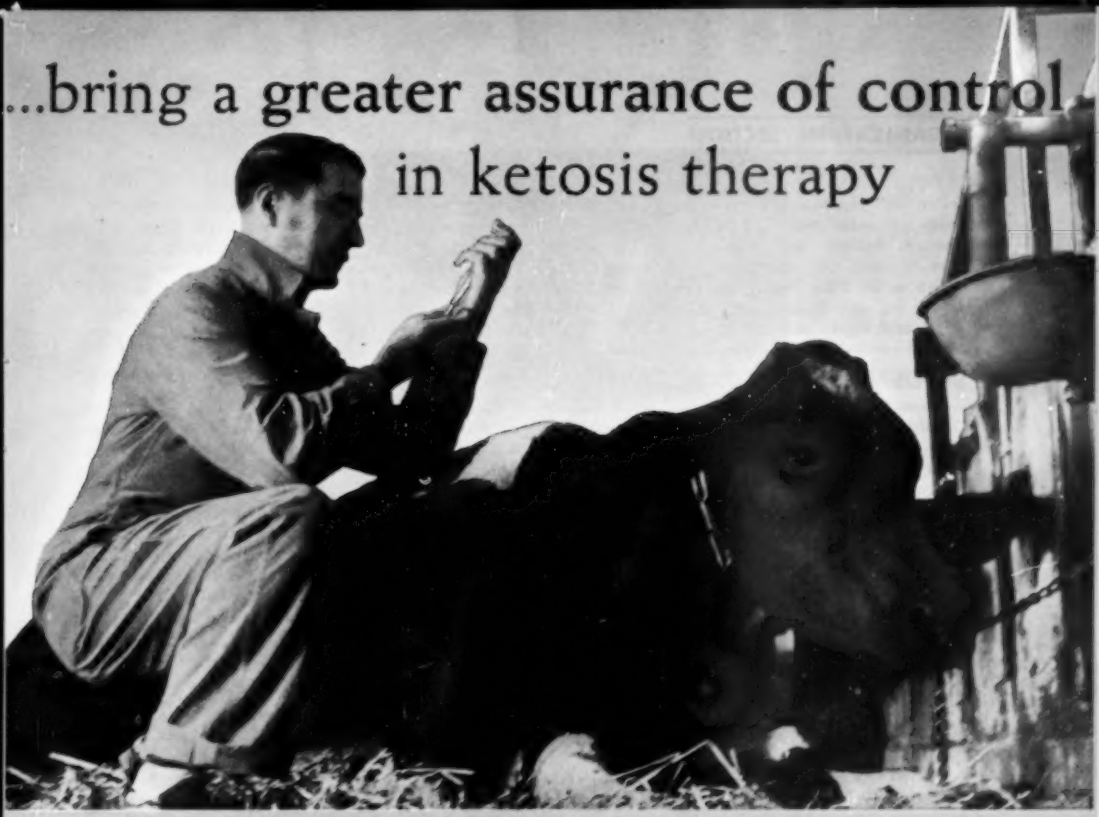

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One is at Hueston Woods in Butler-Preble counties off state road 732. This park also has swimming, fishing, and boating. Lake Hope in Vinton County, off state road 278, has similar facilities as do Pike Park, Pike County, off U. S. highway 50, and Pymatuning, Ashtabula County, off U. S. highways 6 and 322.

STUDENT CHAPTER ACTIVITIES

Oklahoma A. & M. Student Chapter.—Officers of the Oklahoma A. & M. College Student Chapter of the AVMA for the spring semester are Robert Godby, president; Edward McCary, president-elect; Lee Chatham, secretary; Tom Lathan, treasurer; and Bob Spragg, editor. Faculty advisors are R. G. Buckner and J. D. Friend.

Speakers for the year have been E. S. Tierkel, U. S. Public Health Service, Atlanta, Ga.; J. L. Bostwick, Fairview; W. E. Brock, Stillwater; G. T. Eadley, Sulphur; B. J. Myers, Norman; and G. E. Hawley, Charles Pfizer Co., Indianapolis, Ind. Show-dog breeders from Oklahoma City presented a dog show demonstrating the different types and classes of show dogs.

This year, the chapter is producing a yearbook of the members of the School of Veterinary Medicine. The chairman of this project was Joe Jollife who was assisted by two representatives from each class. The book will appear every other year.

s/LEE CHATHAM, *Secretary.*

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Colorado Chapter.—The program of the April 17 meeting of the Colorado Student Chapter of the AVMA included a talk by Dr. M. A. Hammarlund on large animal practice. Dr. Hammarlund is head of the diagnostic laboratory at Colorado State University.

The annual junior and senior banquet on April 27 was followed by the spring dance for the entire student chapter and faculty.

Officers for the spring quarter are: Marvin Beeman, president; Bob Miller, vice-president; C. H. Lehrman, secretary; Kent Herron, treasurer; and Kaye Wilkens, program chairman.

s/C. H. LEHRMAN, *Secretary.*

Aerial view of Columbia Hills Country Club, site of AVMA golf tournament which will be held August 19 in connection with the AVMA convention. The clubhouse and greens 9 and 18 are shown.

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COMING MEETINGS

North Dakota Veterinary Medical Association. Annual meeting. Minot, N. Dak., June 17-18, 1957. Dean Flagg. 202 Teton Ave., Bismarck, N. Dak., secretary.

California State Veterinary Medical Association. Annual convention. Hotel Miramar, Santa Barbara, June 17-19, 1957. Charles S. Travers, 3004 16th St., San Francisco, Calif., secretary.

Vermont Veterinary Medical Association. Annual meeting. The Lodge at Smugglers Notch, Stowe, June 18-19, 1957. Dr. A. E. Janawicz, Department of Agriculture, Montpelier, Secretary.

Idaho Veterinary Medical Association. Summer meeting. Shore Lodge, McCall, June 20-22, 1957. A. P. Schneider, 3025 N. Twenty-Third St., Boise, Idaho, secretary.

South Carolina Associations of Veterinarians. Summer convention. Fort Sumter Hotel, Charleston, June 20-22. Worth Lanier, York, S. Car., secretary.

Alberta Veterinary Medical Association. Annual convention, Lethbridge, Alta., June 21-22, 1957. H. C. Carlson, 9324 148th St., Edmonton, Alta., secretary.

South Carolina Association of Veterinarians. Summer meeting. Fort Sumter Hotel, Charleston, June 21-22, 1957. Worth Lanier, P. O. Box 345, York, S. Car., secretary.

Georgia Veterinary Medical Association. Annual meeting. Athens, June 23-25, 1957. C. C. Rife, 505 Lindbergh Drive, N. E., Atlanta 5, Ga., secretary.

Utah Veterinary Medical Association. Annual meeting. Logan, June 25-26, 1957. J. A. Thomas, P. O. Box 592, Provo, Utah, secretary.

Maritime Veterinary Association. Joint conference. Mount Allison University, Sackville, N. B., June 25-27, 1957. J. F. Frank, Division of Animal Pathology, Box 310, Sackville, N. B., chairman.

North Carolina Veterinary Medical Association. Annual meeting. Grove Park, Asheville, June 25-27, 1957. C. J. Lange, 3741 High Point Rd., Greensboro, N. Car., secretary.

Michigan State Veterinary Medical Association. Annual meeting. Leland Hotel, Detroit, June 26-27, 1957. Paul V. Howard, 4011 Hunsberger, N. E., Grand Rapids 5, Mich., secretary.

Maryland State Veterinary Medical Association. George Washington Hotel, Ocean City, June 27-28, 1957. John D. Gadd, Cockeysville, Md., secretary.

Montana Veterinary Medical Association. Summer meeting. Northern Hotel, Billings, June 28-30, 1957. G. A. Morrison, 316 Central Ave., Great Falls, Mont., secretary.

Symposium on Reproduction and Infertility. Annual meeting. Colorado State University, Fort Collins, July 1-3, 1957. F. X. Gassner, Colorado State University, Fort Collins, Colo., chairman, Committee on Arrangements.

Nebraska State Veterinary Medical Association. Summer meeting. Alliance, July 13-15, 1957. W. T. Spencer, 1250 N. 37th St., Lincoln, Nebr., secretary.

Mississippi State Veterinary Medical Association, Inc. Annual meeting. Hotel Heidelberg, Jackson, July 14-16, 1957. Harvey F. McCrory, Box 536, State College, Miss., secretary.

Kentucky Veterinary Medical Association. Annual meeting. Brown Hotel, Louisville, July 15-16, 1957. Robert H. Singer, 136 Shawnee Place, Lexington, Ky.

Iowa State College. Annual conference for veterinarians. Memorial Union, Ames, July 16-17, 1957. M. S. Hofstad. Veterinary Research Institute, Iowa State College, Ames, program chairman.

Alabama Polytechnic Institute. Annual conference for veterinarians. School of Veterinary Medicine, Auburn, July 21-24, 1957. R. S. Sugg, dean.

Canadian Veterinary Medical Association. Annual meeting. Hotel Georgia, Vancouver, B. C., July 22-24, 1957. James Archibald, Ontario Veterinary College, Guelph, Ont., vice-president.

Colorado Veterinary Medical Association. Annual meeting. LaCourt Hotel, Grand Junction, Aug. 9-10, 1957. G. H. Gilbert, 5500 Wadsworth Blvd., Arvada, Colo., secretary.

American Veterinary Medical Association. Annual meeting. Cleveland Auditorium, Cleveland, Ohio, Aug. 19-22, 1957. J. G. Hardenbergh, 600 S. Michigan Ave., Chicago 5, Ill., executive secretary.

Washington State Veterinary Medical Association. Annual meeting. Monticello Hotel, Longview, Sept. 9-10, 1957. William F. Harris, 1102 E. Main St., Puyallup, Wash., secretary.

New York State Veterinary Medical Society. Annual meeting. Hotel Statler, Buffalo, Sept. 11-13, 1957. M. H. Covert, 138 Inglewood Dr., Rochester 19, N. Y., secretary.

Northern Illinois Veterinary Medical Association. Fall meeting. Rockford, Sept. 18, 1957. J. G. Hardenbergh, 121 Bridge Street, Rockton, Ill., secretary-treasurer.

New England Veterinary Medical Association. Annual meeting. Equinox House, Manchester, Vt., Oct. 6-9, 1957. C. Lawrence Blakely, 180 Longwood Ave., Boston, Mass., secretary.

Purdue University. Annual short course for veterinarians. Purdue University, West Lafayette, Ind., Oct. 9-11, 1957. L. M. Hutchings, secretary.

Florida State Veterinary Medical Association. Annual meeting. Fort Harrison Hotel, Clearwater, Oct. 13-15, 1957. Robert P. Knowles, 2934 N.W. 17th Ave., Miami 37, Fla., secretary.

University of Missouri. Annual short course for graduate veterinarians, Oct. 14-15, 1957, School of Veterinary Medicine, University of Missouri, Columbia. Cecil Elder, chairman.

Texas Veterinary Medical Association. Annual meeting. Baker Hotel, Dallas, Oct. 16-18, 1957. Paul B. Blunt, 712 Maverick Bldg., San Antonio, Texas, secretary.

Eastern Iowa Veterinary Association. Annual meeting. Hotel Sheraton-Montrose, Cedar Rapids, Oct. 17-18, 1957. F. E. Brutsman, Traer, Iowa, secretary.

Southern Veterinary Medical Association. Annual meeting. Hotel Roanoke, Roanoke, Va., Oct. 27-30, 1957. A. A. Husman, P. O. Box 91, Raleigh, N. Car., secretary.

Cornell University. Nutrition conference. Cornell University, Ithaca, N.Y., Oct. 31-Nov. 1, 1957. J. K. Loosli, Stocking Hall, Cornell University, Ithaca, N.Y., chairman.

Animal Care Panel. Annual meeting. Bellevue Hotel, San Francisco, Calif., Nov. 7-9, 1957. R. J. Flynn, Box 299, Lemont, Ill.

Minnesota Veterinary Medical Association. Annual meeting, St. Paul, Jan. 20-22, 1958. B. S. Pomeroy, School of Veterinary Medicine, University of Minnesota, St. Paul 1, Minn.

Kansas Veterinary Medical Association. Annual meeting. Hotel Broadview, Wichita, Feb. 9-11, 1958. K. Maynard Curtz, 5236 Delmar Ave., Kansas City 3, Kan.

Foreign Meetings

British Veterinary Association, Annual congress. University of Cambridge, Cambridge, England, Aug. 25-31, 1957. Mr. F. Knight, 7, Mansfield St., Portland Place, London, W, 1, general secretary.

Regularly Scheduled Meetings

ALABAMA—Central Alabama Veterinary Association, the first Thursday of each month. B. M. Lauderdale, Montgomery, secretary.

Jefferson County Veterinary Medical Association, the second Thursday of each month. S. A. Price, 213 N. 15th St., Birmingham, secretary.

Mobile-Baldwin Veterinary Medical Association, the third Tuesday of each month. W. David Gross, 771 Holcombe Ave., Mobile, Ala., secretary.

ARIZONA—Central Arizona Veterinary Medical Association, the second Tuesday of each month. Keith T. Maddy, Phoenix, Ariz., secretary.

Southern Arizona Veterinary Medical Association, the third Wednesday of each month at 7:30 p.m. E. T. Anderson, Rt. 2. Box 697, Tucson, Ariz., secretary.

CALIFORNIA—Alameda Contra Costa Veterinary Medical Association, last Wednesday of each month. Leo Goldston, 3793 Broadway, Oakland 11, Calif., secretary.

Bay Counties Veterinary Medical Association, the second Tuesday of each month. Maurice L. Boevers, 3394 Mt. Diablo Blvd., Lafayette, Calif., secretary.

Central California Veterinary Medical Association, the fourth Tuesday of each month. R. B. Barsaleau, 2333 E. Mineral King, Visalia, Calif., secretary.

Kern County Veterinary Medical Association, the first Thursday evening of each month. A. L. Irwin, 301 Taft Highway, Bakersfield, Calif., secretary.

Mid-Coast Veterinary Medical Association, the first Thursday of every even month. W. H. Rockey, P. O. Box 121, San Luis Obispo, Calif., secretary.

Monterey Bay Area Veterinary Medical Association, the third Wednesday of each month. Lewis J. Campbell, 90 Corral de Tierra, Salinas, Calif., secretary.

North San Joaquin Valley Veterinary Medical Association, the fourth Wednesday of each month at the Hotel Covell, in Modesto, Calif. Lyle A. Baker, Turlock, Calif., secretary.

Orange Belt Veterinary Medical Association, the second Monday of each month. Chester A. Maeda, 766 E. Highland Ave., San Bernardino, Calif., secretary.

Orange County Veterinary Medical Association, the third Thursday of each month. Donald E. Lind, 2643 N. Main St., Santa Ana, Calif., secretary.

Peninsula Veterinary Medical Association, the third Monday of each month. R. C. Lawson, 4040 El Camino, Palo Alto, Calif., secretary.

Redwood Empire Veterinary Medical Association, the third Thursday of each month. Robert E. Clark, Napa, Calif., secretary.

Sacramento Valley Veterinary Medical Association, the second Wednesday of each month. W. E. Steinmetz, 4227 Freeport Blvd., Sacramento, Calif., secretary.

San Diego County Veterinary Medical Association, the fourth Tuesday of each month. H. R. Rossoll, 1795 Moore St., San Diego, Calif., secretary.

San Fernando Valley Veterinary Medical Association, the second Friday of each month at the Casa Escobar Restaurant in Studio City. John Chudacoff, 7912 Sepulveda Blvd., Van Nuys, secretary.

Santa Clara Valley Veterinary Association, the fourth Tuesday of each month. Kay Beulley, N. Fourth and Gish Rd., San Jose, Calif., secretary.

Southern California Veterinary Medical Association, the last Wednesday of each month. Don Mahan, 1919 Wilshire Blvd., Los Angeles 57, Calif., executive secretary.

Tulare County Veterinarians, the second Thursday of each month. R. B. Barsaleau, 2333 E. Mineral King, Visalia, Calif., secretary.

COLORADO—Denver Area Veterinary Society, the fourth Tuesday of every month. Richard C. Tolley, 5060 S. Broadway St., Englewood, Colo., secretary.

Northern Colorado Veterinary Medical Society, the first Monday of each month. M. A. Hammarlund, School of

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REFERENCES: 1. Bull, W. S.: N. Amer. Vet., In press. 2. Henry, R. T., and Blackburn, E. G.: Vet. Med., In press.

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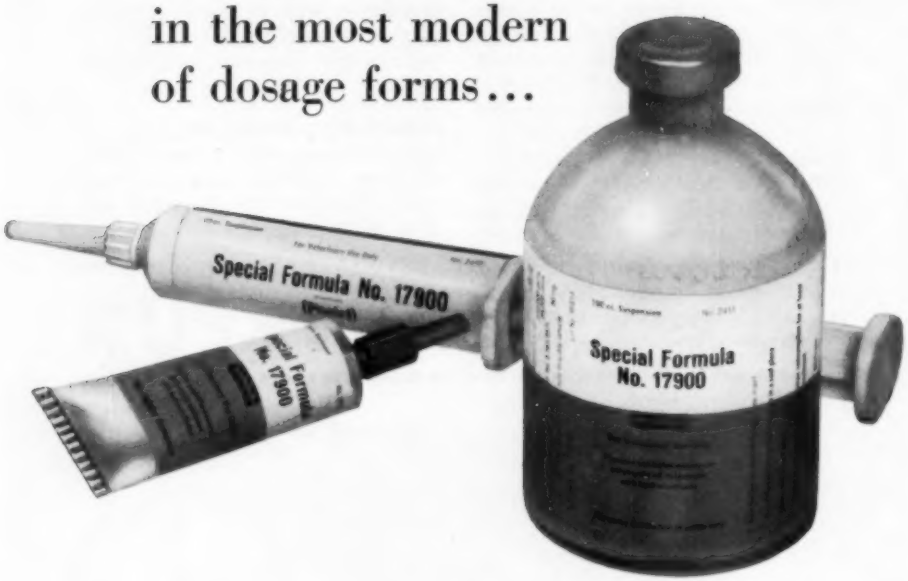

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Veterinary Medicine, Colorado A. & M. College, Fort Collins, Colo., secretary.

DELAWARE—New Castle County Veterinary Association, the first Tuesday of each month at 9:00 p.m. in the Hotel Rodney, Wilmington, Del. E. J. Hathaway, Clifton Park Manor, Apt. 73-5, Wilmington 2, Del., secretary.

FLORIDA—Central Florida Veterinary Medical Association, the first Tuesday of each month, time and place specified monthly. Jack H. McElyer, 5925 Edgewater Drive, Orlando, Fla., secretary.

Jacksonville Veterinary Medical Association, the first Thursday of every month. Dodsons Restaurant. P. S. Roy, 4443 Atlantic Blvd., Jacksonville, Fla., secretary.

Northwest Florida Veterinary Medical Society, third Wednesday of each month, time and place specified monthly. T. R. Geci, 108B Catherine Ave., Pensacola, Fla., secretary.

Palm Beach Veterinary Society, the last Thursday of each month in the county office building at 810 Datura St., West Palm Beach. J. J. McCarthy, 500-25th Street, West Palm Beach, Fla., secretary.

Ridge Veterinary Medical Association, the fourth Thursday of each month in Bartow, Fla. Paul J. Myers, Winter Haven, Fla., secretary.

South Florida Veterinary Society, the third Tuesday of each month, at the Seven Seas Restaurant, Miami, Fla. E. D. Stoddard, 6432 S. W. 8th St., Miami, Fla., secretary.

Suwannee Valley Veterinary Association, the fourth Tuesday of each month, Hotel Thomas, Gainesville. W. B. Martin, Jr., 3002 N. W. 6th St., Gainesville, Fla., secretary.

Volusia County Veterinary Medical Association, the fourth Thursday of each month. A. E. Hixon, 131 Mary St., Daytona Beach, Fla., secretary.

GEORGIA—Atlanta Veterinary Society, the second Tuesday of every month at the Elks Home on Peachtree St., Atlanta, Ga. J. L. Christopher, Smyrna, Ga., secretary.

ILLINOIS—Chicago Veterinary Medical Association, the second Tuesday of each month. Mark E. Davenport, Jr., 215 S. Edgewood Ave., LaGrange, Ill., secretary.

Eastern Illinois Veterinary Medical Association, the first Thursday of March, June, September, and December. A one-day clinic is held in May. H. S. Bryan, College of Veterinary Medicine, University of Illinois, Urbana, secretary.

INDIANA—Central Indiana Veterinary Medical Association, the second Wednesday of each month. Peter Johnson, Jr., 4410 N. Keystone Ave., Indianapolis 5, secretary.

Michiana Veterinary Medical Association, the second Thursday of every month except July and December, at the Hotel LaSalle, South Bend, Ind. J. M. Carter, 3421 S. Main St., Elkhart, Ind., secretary.

Tenth District Veterinary Medical Association, the third Thursday of each month. J. S. Baker, P. O. Box 52, Pendleton, Ind., secretary.

IOWA—Cedar Valley Veterinary Association, the second Monday of each month, except January, July, August, and October, at Black's Tea Room, Waterloo, Iowa. H. V. Henderson, Reinbeck, Iowa, secretary.

Coon Valley Veterinary Association, the second Wednesday of each month, September through May, at the Bradford Hotel, Storm Lake, Iowa. D. I. Lee, Sac City, Iowa, secretary.

East Central Iowa Veterinary Medical Society, the second Tuesday of every month. Dr. W. T. Rugger, Oxford, secretary.

Fayette County Veterinary Association, the third Tuesday of each month, except in July and August, at Pa and Ma's Restaurant, West Union, Iowa. Donald E. Moore, Box 178, Decorah, Iowa, secretary.

Northeast Iowa-Southern Minnesota Veterinary Association, the first Tuesday of February, May, August, and November at the Winneslick Hotel, Decorah, Iowa. 6:30 p.m. Donald E. Moore, Box 178, Decorah, Iowa secretary.

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KENTUCKY—Central Kentucky Veterinary Medical Association, the first Wednesday of each month. L. S. Shirrell, Versailles Rd., Frankfort, secretary.

Jefferson County Veterinary Society of Kentucky, Inc., the first Wednesday evening of each month in Louisville or within a radius of 50 miles. W. E. Bewley, P.O. Box "H," Crestwood, secretary.

MARYLAND—Baltimore City Veterinary Medical Association, the second Thursday of each month, September through May (except December), at 9:00 p.m. at the Park Plaza Hotel, Charles and Madison St., Baltimore, Md. Harry L. Schultz, Jr., 9011 Harford Rd., Baltimore, Md., secretary.

MICHIGAN—Mid-State Veterinary Medical Association, the fourth Thursday of each month with the exception of November and December. Robert E. Kader, 5034 Armstrong Rd., Lansing 17, Mich., secretary.

Saginaw Valley Veterinary Medical Association, the last Wednesday of each month. S. Correll, Rt. 1, Midland, Mich., secretary.

Southeastern Veterinary Medical Association, the fourth Wednesday of every month, September through May. Gilbert Meyer, 14003 E. Seven Mile Rd., Detroit 5, Mich., secretary.

MISSOURI—Greater St. Louis Veterinary Medical Association, the first Friday of the month (except July and August) at the Sheraton Hotel, Spring Ave. and Lindell Blvd. Allen B. Shopmaker, 136 N. Meramec, Clayton 5, Mo., secretary.

Kansas City Small Animal Hospital Association, the first Monday of each month, at alternating hospitals. W. F. Noland, 7504 Metcalf, Overland Park, Kan., secretary.

Kansas City Veterinary Medical Association, the third Tuesday of each month at Exchange Hall, ninth floor, Livestock Exchange Bldg., 1600 Genessee St., Kansas City, Mo. Busch Meredith, 800 Woodswether Rd., Kansas City 5, Mo., secretary.

NEW JERSEY—Central New Jersey Veterinary Medical Association, the second Thursday of November, January, March, and May at Old Hights Inn, Hightstown, N. J. David C. Tudor, Cranbury, N. J., secretary.

Metropolitan New Jersey Veterinary Medical Association, the third Wednesday evening of each month from October through April at the Academy of Medicine, 91 Lincoln Park South, Newark, N. J. Myron S. Arlein, 2172 Milburn Ave., Maplewood, N. J., secretary.

Northern New Jersey Veterinary Association, the fourth Tuesday of each month at the Casa Mana in Teaneck. James R. Tanzola, Upper Saddle River, secretary.

Northwest Jersey Veterinary Society, the third Wednesday of every odd month. G. R. Muller, 43 Church St., Lambertville, N. J., secretary.

Southern New Jersey Veterinary Medical Association, the third Tuesday of each month at the Collingswood Veterinary Hospital, Collingswood. W. E. Snyder, E. Kings Highway and Munn Ave., Haddonfield, secretary.

NEW YORK—New York City, Inc., Veterinary Medical Association of, the first Wednesday of each month at the New York Academy of Sciences, 2 East 63rd St., New York City. C. E. DeCamp, 43 West 61st St., New York 23, N. Y., secretary.

New York State Veterinary College. Annual conference for veterinarians. Cornell University, Ithaca. W. A. Hagan, New York State Veterinary College, Cornell University, Ithaca, N. Y., dean.

Monroe County Veterinary Medical Association, the first Thursday of even-numbered months except August. Irwin Bircher, 50 University Ave., Rochester, N. Y., secretary.

NORTH CAROLINA—Central Carolina Veterinary Medical Association, the second Wednesday of each month at 7:00 p.m. in the O'Henry Hotel, Greensboro. Joseph A. Lombardo, 411 Woodlawn Ave., Greensboro, secretary.

Eastern North Carolina Veterinary Medical Association, the first Friday of each month, time and place specified

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Piedmont Veterinary Medical Association, the last Friday of each month. John G. Martin, Boone, N. Car., secretary.

Twin Carolinas Veterinary Medical Association, the third Thursday of each month in the Orange Bowl Restaurant, Rockingham, N. Car., at 7:30 p.m. James R. Burgess, Rockingham, N. Car., secretary.

OHIO—Cuyahoga County Veterinary Medical Association, the first Wednesday of each month, September through May (except January), at 9:00 p.m. at the Carter Hotel, Cleveland, Ohio. Ed. R. Jacobs, 5522 Pearl Rd., Cleveland, Ohio, secretary.

OKLAHOMA—Oklahoma County Veterinary Medical Association, the second Wednesday of every month, 7:30 p.m., Patrick's Foods Cafe, 1016 N.W. 23rd St., Oklahoma City. Forrest H. Stockton, 2716 S.W. 29th St., Oklahoma City, Okla., secretary.

Tulsa Veterinary Medical Association, the third Thursday of each month in Directors' Parlor of the Brookside State Bank, Tulsa, Okla. Don L. Hohmann, 538 S. Madison St., Tulsa, Okla., secretary.

PENNSYLVANIA—Keystone Veterinary Medical Association, the fourth Wednesday of each month at the University of Pennsylvania School of Veterinary Medicine, 39th and Woodland Ave., Philadelphia 4, Pa. Raymond C. Snyder, 39th and Woodland Ave., Philadelphia 4, Pa., secretary.

SOUTH CAROLINA—Piedmont Veterinary Medical Association, the third Wednesday of each month at the Fairforest Hotel, Union, S. Car. Worth Lanier, York, S. Car., secretary.

TEXAS—Coastal Bend Veterinary Association, the second Wednesday of each month. J. Marvin Prewitt, 4141 Lexington Blvd., Corpus Christi, Texas, secretary.

VIRGINIA—Central Virginia Veterinarians' Association, the third Thursday of each month at the William Byrd Hotel in Richmond at 8:00 p.m. M. R. Levy, 312 W. Cary St., Richmond 20, Va., secretary.

Northern Virginia Veterinary Society, the second Wednesday of every third month. Meeting place announced by letter. H. C. Newman, Box 145, Merrifield, secretary.

Southwest Virginia Veterinary Medical Association, the first Thursday of each month. I. D. Wilson, Blacksburg, secretary.

WASHINGTON—Seattle Veterinary Medical Association, the third Monday of each month, Magnolia American Legion Hall, 2870 32nd W., Seattle, Wash. William S. Green, 9637 S. E. 36th, Mercer Island, Wash., secretary.

South Puget Sound Veterinary Association, the second Thursday of each month except July and August. O. L. Bailey, P. O. Box 906, Olympia, Wash., secretary.

WEST VIRGINIA—Kyowva (Ky., Ohio, W. Va.) Veterinary Medical Association, the second Thursday of each month in the Hotel Prichard, Huntington, W. Va., at 8:30 p.m. Harry J. Fallon, 200 5th St., W. Huntington, W. Va., secretary.

Central Wisconsin Veterinary Medical Association, the second Tuesday of each quarter (March, June, Sept., Dec.). R. J. O'Hern, P. O. Box 617, Cumberland, Wis., secretary.

Dane County Veterinary Medical Association, the second Thursday of each month. Dr. E. P. Pope, 409 Farley Ave., Madison, Wis., secretary.

WISCONSIN—Milwaukee Veterinary Medical Association, the third Tuesday of each month, at the Half-Way House, Blue Mound Rd. George F. Lynch, 201 West Devon St., Milwaukee 17, Wis., secretary.

Northeastern Wisconsin Veterinary Medical Association, the third Wednesday in April. William Madison, 218 E. Washington St., Appleton, Wis., secretary.

Rock Valley Veterinary Medical Association, the first Wednesday of each month. W. E. Lyle, P. O. Box 107, Deerfield, Wis., secretary.

Southeastern Veterinary Medical Association, the third Thursday of each month. John R. Curtis, 419 Cook St., Portage, Wis., secretary.

Wisconsin Valley Veterinary Medical Association, the second Tuesday of every other month. E. S. Scobell, Rt. 2, Wausau, Wis., secretary.

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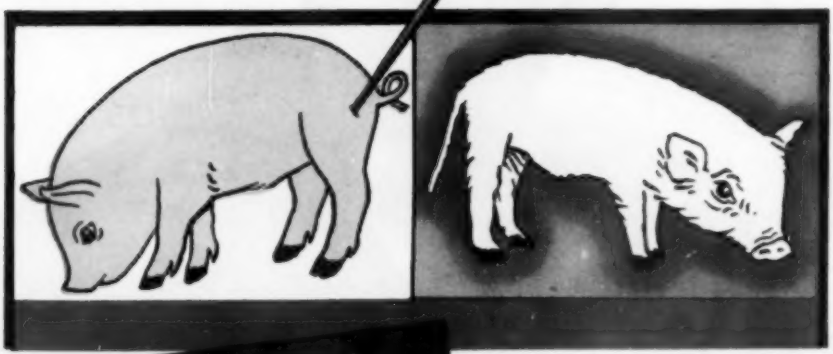

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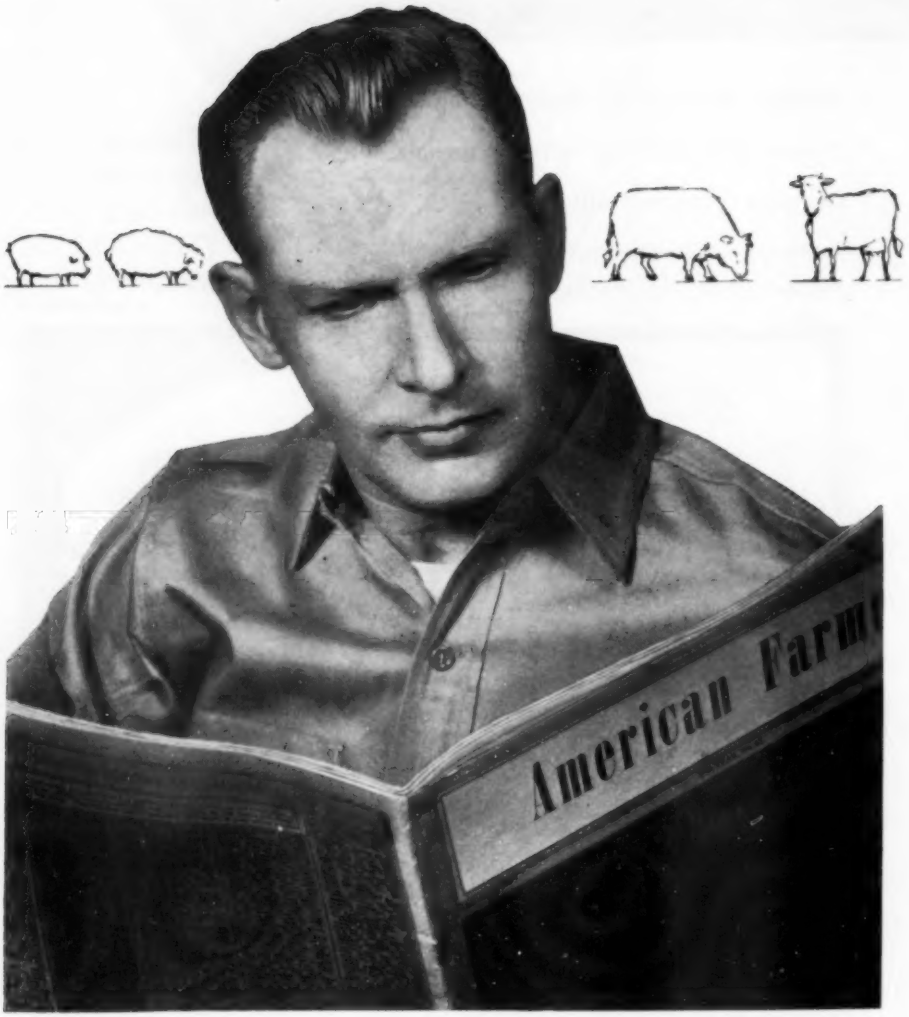

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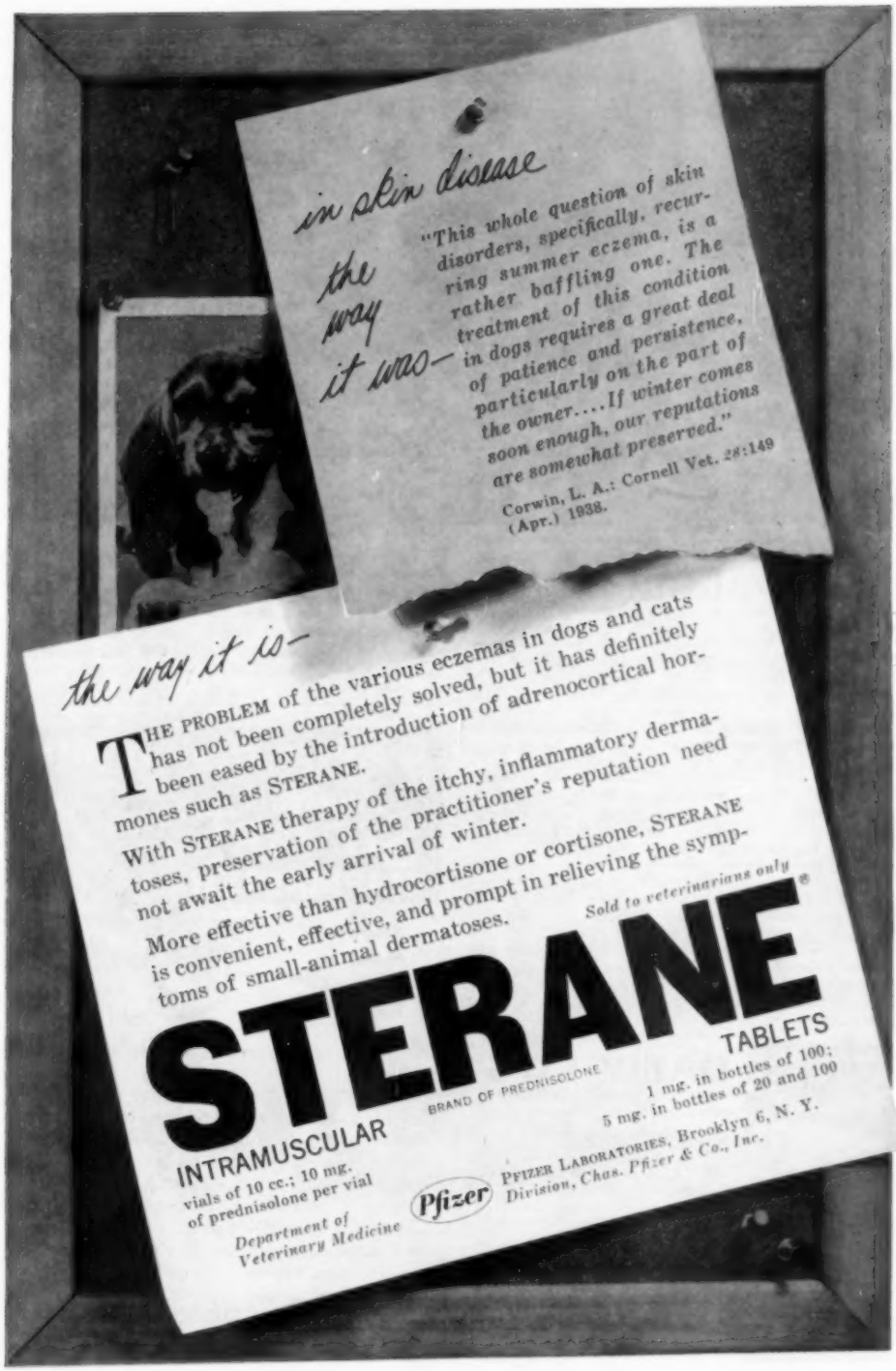
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REFERENCES: 1. Mosier, J. E.: Vet. M. 50:605 (Nov.) 1955. 2. Selloff, G. B.: Calif. Vet. 9:27 (Sept.-Oct.) 1956. 3. Pollock, S.: J. Am. Vet. M. Ass. 129:274 (Sept.) 1956.

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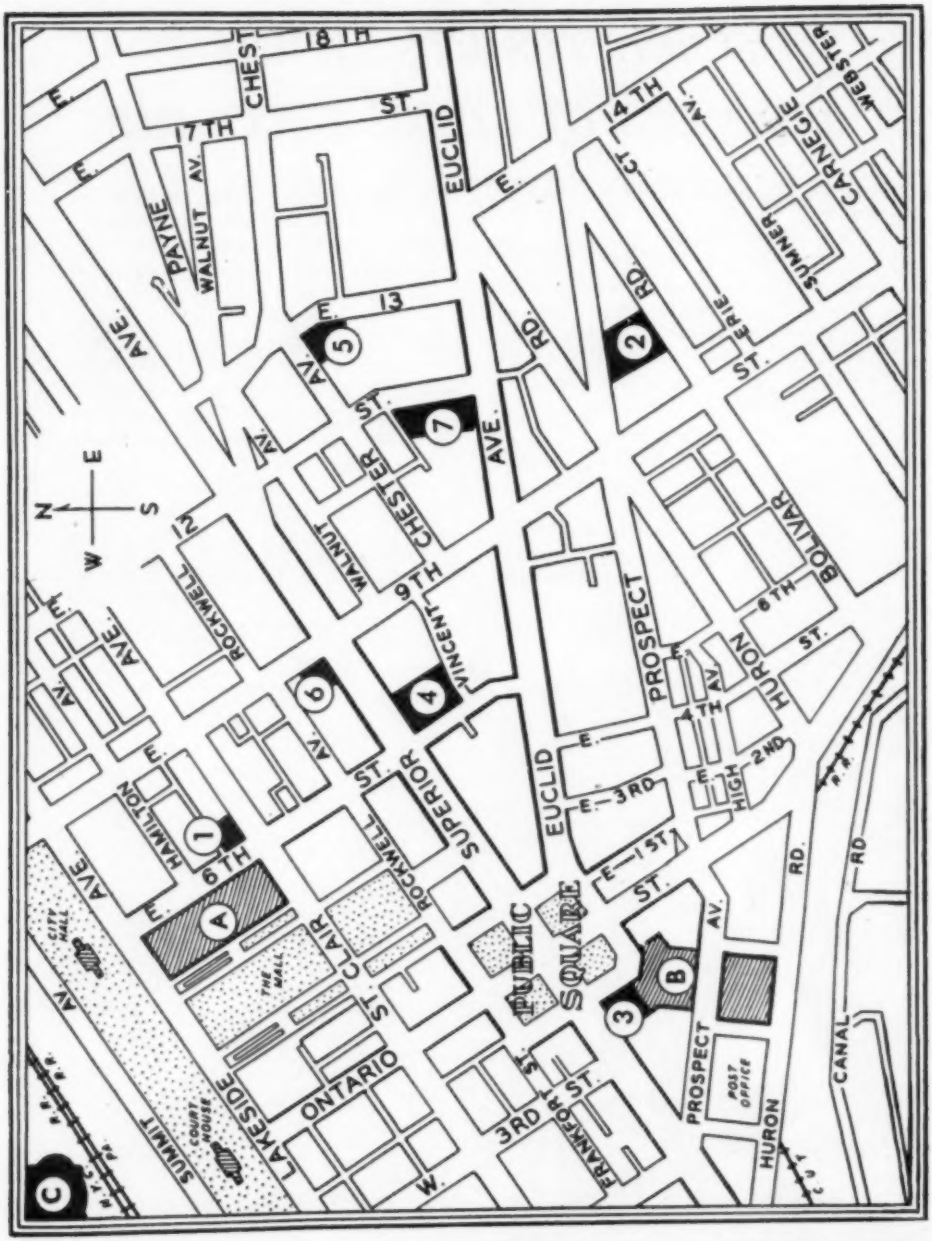

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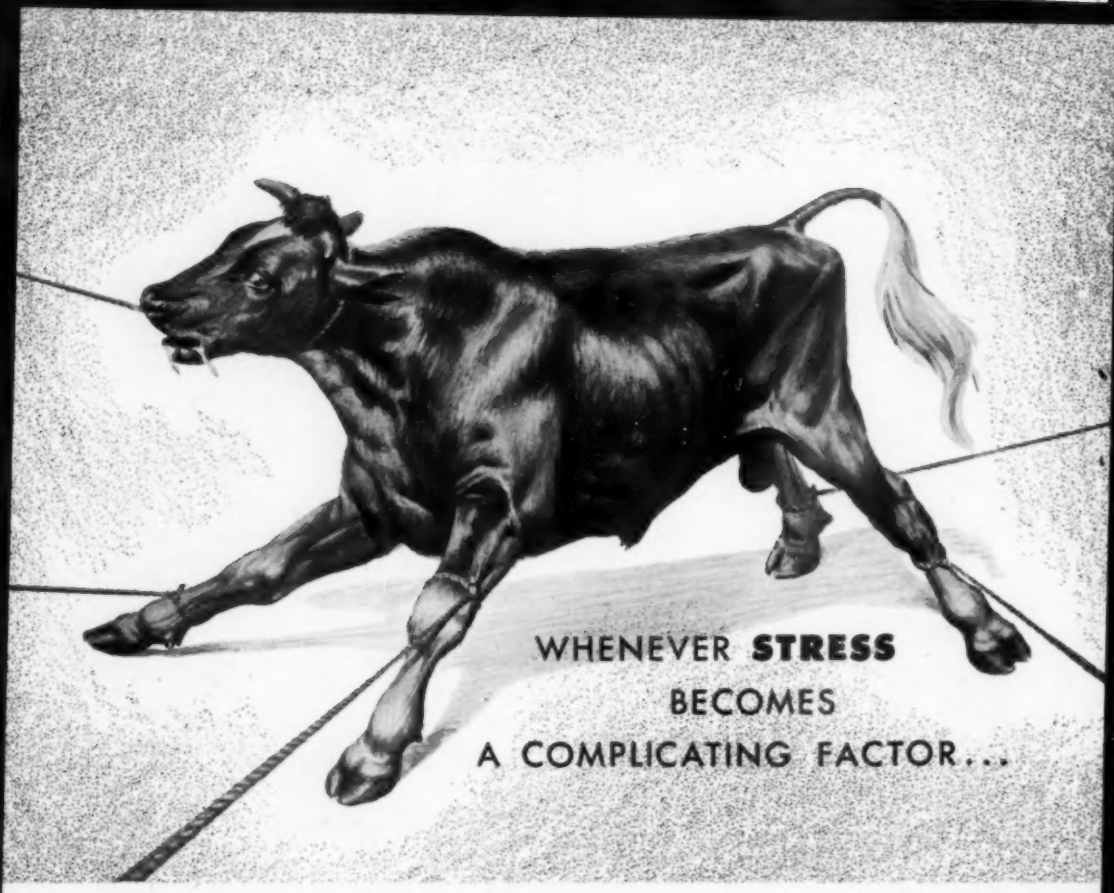

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